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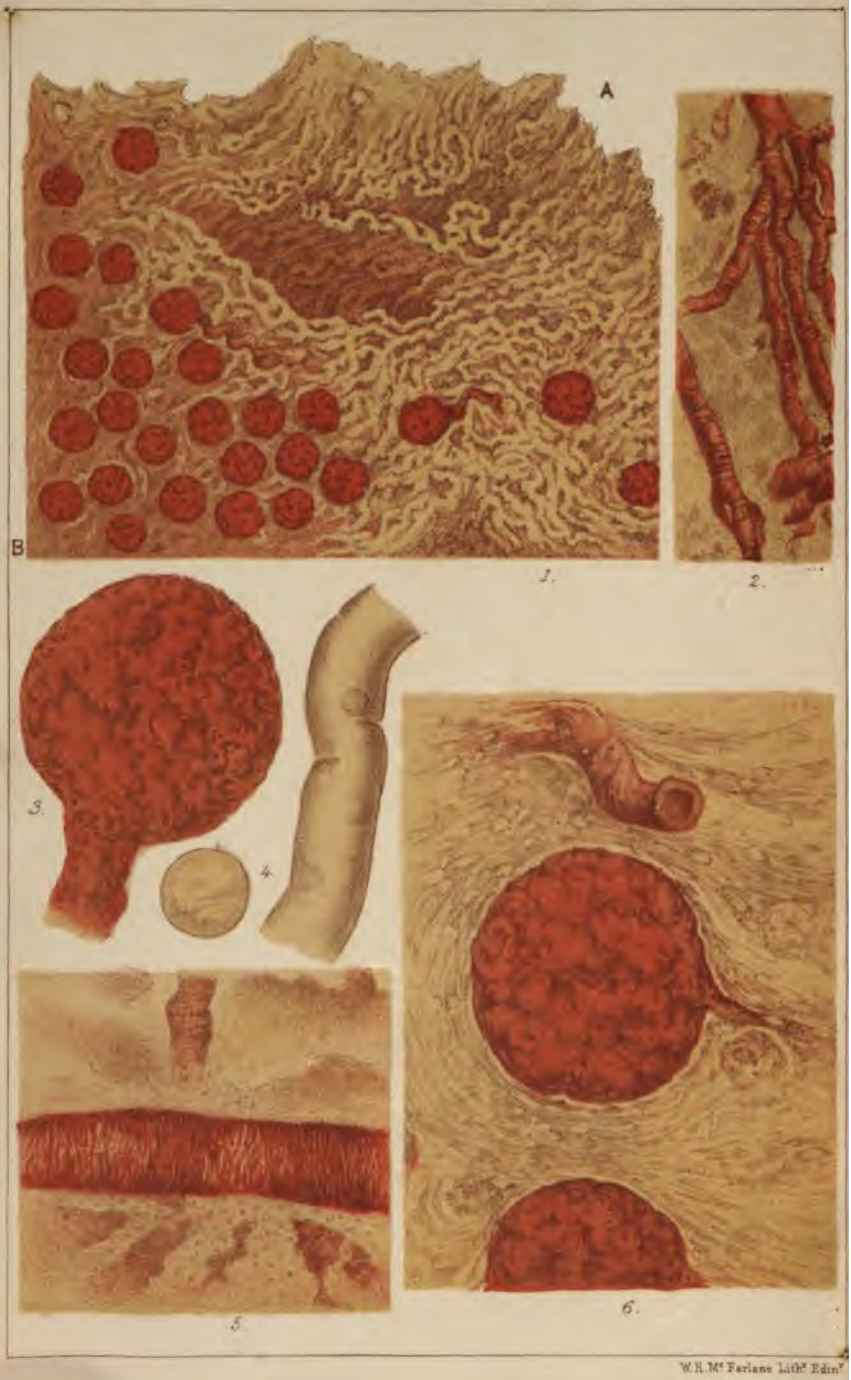

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A PRACTICAL TREATISE

ON

BRIGHT'S DISEASES OF THE KIDNEYS.

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BY

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OF EDINBURGH.

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BY

T. GRAINGER STEWART, M.D., F.R.S.E.,

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TO

ROBERT CHRISTISON, M.D., D.C.L.,

VICE-PRESIDENT OF THE ROYAL SOCIETY,

PROFESSOR OF MATERIA MEDICA IN THE UNIVERSITY OF EDINBURGH,

ONE OF THE FIRST TO APPRECIATE THE DISCOVERIES OF BRIGHT,

AND TO RENDER THEM MORE ILLUSTRIOUS

BY ESTABLISHING THEIR ACCURACY,

AND ELUCIDATING, BY MEANS OF ABLE AND ORIGINAL

OBSERVATION AND RESEARCH,

MANY POINTS IN THE NATURAL HISTORY AND TREATMENT OF

RENAL DISEASES,

THIS BOOK IS RESPECTFULLY

DEDICATED.

PREFACE.

In the following pages I have endeavoured to embody the views which I have been led to entertain in regard to the renal affections, commonly included under the term Bright's Disease or Albuminuria. These views have already been in part laid before the profession in occasional papers in the medical journals, but it seemed desirable to collect and rearrange them for practical convenience.

The title "Bright's Diseases" has been chosen because it is desirable to preserve the memory of the illustrious discoverer in connection with his work, and because we can no longer speak of one disease discovered by him, but must recognise several distinct diseases.

I have to record my very grateful thanks to my colleagues in the Royal Infirmary for the permission they have always given to observe and record cases in which I was specially interested. All the cases not under my own care which appear in this work, have, with two exceptions, been already published.

I have also to express my obligations to my friends, Drs Argyll Robertson, Ireland, Gamgee, and Rutherford, who have favoured me with much valuable assistance and advice.

Three subjects of special pathological interest are considered in Supplementary Chapters.

The Plates have been prepared by Messrs Schenck & Macfarlane, from drawings made from nature by Mr Neil Stewart, Mr Weisse, and myself.

25 QUEEN STREET, EDINBURGH,
August, 1868.

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A PRACTICAL TREATISE

ON

BRIGHT'S DISEASES OF THE KIDNEYS.

CHAPTER I.

INTRODUCTION AND CLASSIFICATION.

In the year 1827, Dr Bright published, in his select report of medical cases, an account of some of the appearances observable on the examination of cases terminating in dropsical effusion, and then, for the first time, specially directed the attention of the profession to certain morbid conditions of the kidney, which frequently co-exist with the symptom in question. He showed, further, that a secretion of albuminous urine frequently co-existed with the other conditions. Dr Blackall had previously shown that a relationship exists between dropsy and albuminuria, but had not observed the morbid conditions of the kidney, then first described by Dr Bright.* Dr Bright further figured, in a series of plates unsurpassed in

* Report of Medical Cases, by Richard Bright, London, 1827.

medical literature, some of the conditions of the kidney which he had observed. To the disease thus described, the name "Morbus Brightii," or "Bright's disease," was applied, and we think it convenient still to retain the term, although it must now be understood to include several different morbid conditions of the kidney, with corresponding symptoms and complications; for this reason, I prefer to speak of Bright's diseases.

It is evidently difficult, or almost impossible, accurately to define the term Bright's disease, but it may be generally described as including the diseases of the kidney which are accompanied, at one stage or other of their course, by albuminuria or dropsy, or by both.

Dr Christison was one of the first to recognise the value of Bright's observations, and to confirm them by his own; and in his work on the granular degeneration of the kidneys,* published in 1839, he contributed much to our knowledge of the subject. I do not propose to enter upon the somewhat unprofitable topic of the gradual elucidation of this subject, but shall pass on to treat of it as I believe it is at present best understood.

In his cellular pathology, Professor Virchow states,† that as there are three main elements in the kidney, viz., tubules, vessels, and interstitial tissue, so there are three forms of Bright's disease, one originating in each of the elements. Thus, in the tubules we have

* On Granular Degeneration of the Kidneys, &c., by Robert Christison, M.D., F.R.S.E., &c., Edinburgh, 1839.

† Virchow's Cellular Pathology, translated by Dr Chance, p. 380.

what he terms Parenchymatous Nephritis; in the vessels, Amyloid Degeneration; in the interstitial tissue, Cirrhosis. He justly remarks, that these three different forms by no means always appear unmixed, but that, on the contrary, frequently two, and sometimes all of them exist in the same kidney. His classification we believe to be correct; we shall therefore retain it, with some slight modifications to be presently described.

It may be well, before giving a detailed account of these forms, to indicate and briefly consider the classification adopted by a few recent authorities.

One of the best recent German writers on this subject, Dr Rosenstein,* of Berlin, conceives that under the term Bright's disease four morbid conditions are included, viz.:—1st, Congestion from obstructed circulation; 2nd, Catarrhal nephritis; 3rd, Diffuse nephritis; 4th, Amyloid degeneration. The two former he regards as examples of slight parenchymatous inflammation (Virchow); the two latter, he says, are examples of severe parenchymatous changes. We differ from this author, inasmuch as we think a distinction between his first three forms unnecessary, and that his fourth form has no essential connection with inflammation of the tubules; and further, we recognise an important form of which he makes no mention, but which is referred to by Virchow, and has been the subject of careful study in this country, viz., the affection of the interstitial tissue, the disease commonly called contracting, cirrhotic, or gouty kidney.

* Die Pathologie und Therapie der Nierenkrankheiten, von Dr S. Rosenstein, Berlin, 1863.

It may be necessary to say a few words in vindication of these opinions, and in explanation of Dr Rosenstein's views. First, then, with regard to his subdivisions of the parenchymatous form. His first variety is, *Bright's disease from congestion, a result of embarrassed circulation.* He agrees with Traube in regarding this as an "affectio sui generis," and not identical with the first stage of diffuse nephritis. The amount of urine is diminished, while the solids are natural. The specific gravity is therefore high, and sediments are readily formed. From pressure on the veins, albumen appears in the urine, and this comes and goes according to the degree of embarrassment of the circulation. In cases of long standing, the anatomical characters of the kidneys are as follows:—Their volume is increased, rarely somewhat diminished, the consistence dense, the capsule easily separable, the surface mostly smooth, sometimes with little patches of depression; on section the cortical substance is usually thick, reddish, or grey, the cones are red, but at the apices generally pale. On microscopic examination, the malpighian bodies appear mostly natural, sometimes a little atrophied; the connective tissue is occasionally found increased, its nuclei abnormally numerous; the epithelium is granular, sometimes fatty, frequently contains pigment; while the veins, particularly the small ones, are much dilated. Such conditions occur occasionally in heart disease, pulmonary affections, and in pregnancy. In a great majority of cases of this kind, however, I have found the kidney not inflamed, the symptoms depending entirely upon hindrance to the circulation. It is true

that in cases of some standing an induration of the organ occurs, and that in a very small proportion of instances a true inflammatory action co-exists with, and is doubtless aggravated by, but does not depend upon, the cardiac or vascular obstruction. Thus I incline to put this form out of the category of Bright's disease altogether.

With regard to his second variety, the *catarrhal nephritis*, I think that it is not properly distinguishable from his diffuse form, and that it occurs in a much greater variety of cases than has been usually supposed. The following is an abstract of the account of it given by Rosenstein :—The pathological appearances are not such as to attract attention; the organ is of normal size, or slightly swollen, in the more severe cases congested, and with ecchymotic spots scattered throughout its substance; the affection begins at the apices of the pyramids, which are at first congested, but afterwards pale, the pallor depending upon the accumulation of swollen epithelium within the tubules. The cortical substance, and especially the parts of the tubules next to the malpighian bodies, often remain free from disease. The stroma, in many cases, is increased in volume. The symptoms, also, are by no means prominent, consisting merely of the occurrence of albumen, mucus, and tube-casts in the otherwise natural urine; the casts are epithelial and hyaline, and sometimes contain blood corpuscles, pigment-granules, and crystals of oxalate of lime. The malady originates under a considerable variety of circumstances,—from exposure to cold, from extension of inflammation from other parts of the urinary tract, from the presence in

the blood of irritating substances, such as canthrides and cubebs, from fever poisons, and in connection with cholera. Such is, in outline, Rosenstein's account of catarrhal nephritis. It appears to me, that in whatever aspect we regard it, it constitutes but a variety of the inflammatory affection. As to its morbid anatomy, it essentially corresponds with ordinary inflammation, consisting, as it does, of an infiltration and cloudy swelling of the epithelium of the tubules. It is true that the number and extent of tubules affected differs from what we see in cases of typical inflammatory Bright's disease, and that the rapidity of the changes induced are also various; but, in acknowledged diffuse inflammation, as well as what is called catarrh, the greatest variety exists, and it is impossible, at any point, to draw a line of demarcation between the two affections. Again, as to symptoms, they are exactly such as would be met with in a mild case, or in an early stage of inflammatory Bright's disease. And, lastly, with regard to the causes; it is obvious that most of those enumerated by Rosenstein, if intensely or continuously applied, would, unquestionably, lead to what all recognise as inflammation of the kidneys. Thus, in respect of morbid anatomy, symptoms, and causes, this affection seems to be undistinguishable from inflammatory Bright's disease.

Of Rosenstein's *diffuse nephritis*, it is unnecessary to say more than that it corresponds to Virchow's parenchymatous form, and to what I shall term the inflammatory form of Bright's disease. He rightly distinguishes the *amyloid affection*; but, as has been

already said, takes no notice of a form which Virchow had recognised, and which, though more rare, unquestionably does occur, the *contracting or cirrhotic kidney.*

As examples of the views of classification prevalent among British authors, I select those of Professors Bennett and Aitken, of Dr Roberts, and of Dr Dickinson. The first* of these authorities describes three forms—1st, Inflammatory, which may be acute or chronic; 2nd, The waxy degeneration; and 3rd, The fatty form. Dr Aitken† distinguishes two varieties of inflammation of the kidney—viz., the parenchymatous, in which the tubules, and the interstitial, in which the stroma, is affected; and, in another part of his work, he mentions several other forms of disease—viz., the large white kidney, the small contracted kidney, mixed forms, fatty, and amyloid degenerations.

Dr Roberts,‡ in his valuable volume on urinary and renal diseases, distinguishes between acute Bright's disease and chronic, and divides the latter class into three varieties—viz., the smooth white kidney, the granular red kidney, and lardaceous or waxy kidney.

Dr Dickinson,§ in his work published since the greater part of this treatise was written, describes three forms of Bright's disease, viz:—1. Tubal nephritis, which never passes on to present the atrophied

* The Principles and Practice of Medicine, by J. Hughes Bennett, M.D., Edinburgh, 1865.

† The Science and Practice of Medicine, by William Aitken, M.D., London, 1863.

‡ A Practical Treatise on Urinary and Renal Diseases, &c., by William Roberts, M.D., London, 1865.

§ On the Pathology and Treatment of Albuminuria, by W. H. Dickinson, M.D. Cantab.

granular rough appearance. 2. Granular degeneration. 3. The depurative, waxy, or amyloid form.

Thus all these writers recognise clearly the more important varieties of the malady; but, well as they have described the diseases, I hope, in the following pages, to bring out certain points in the pathology and clinical history which they have overlooked, but which appear to me of considerable importance. I shall defer the discussion of these points until we have to deal with them in detail, and shall now only indicate the classification which is to be the basis of the present treatise.

Bright's diseases may, I think, be classified as follows :—

1. *The inflammatory form,* of which there are three stages—

a That of inflammation.
b ,, fatty transformation.
c ,, atrophy.

2. *The waxy or amyloid form,* of which also there are three stages—

a That of degeneration of vessels.
b ,, secondary changes in the tubes.
c ,, atrophy.

3. *The cirrhotic, contracting, or gouty form.*

Each of these we shall consider in the sequel.

PLATE I.

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W. H. Mc Farlane, Lith.r Ed.

CHAPTER II.

THE INFLAMMATORY FORM.

Morbid Anatomy.

The inflammatory form of Bright's disease is an affection sometimes of short, sometimes of long, duration. When it runs through its whole course, the kidney undergoes a series of changes, which may, for convenience in description, be divided into three stages, each characterised by very distinct anatomical characters, viz.—1st, that of inflammation; 2nd, that of fatty transformation; and 3rd, that of atrophy. We shall now describe these in their order.

1st, *The stage of inflammation.*—The organ is of the natural size, or somewhat larger; its capsule is unaltered, and strips off readily; its surface is smooth, more or less congested, often pink, it is sometimes of a dark purplish colour, sometimes mottled, pale, and purple. On section, the cortical substance is relatively somewhat increased in volume. It is often congested, the malpighian bodies standing out prominently from the surrounding tissue, the congested vessels separated by a varying amount of white somewhat opaque deposit, composed of the altered cubules, to be presently described. The vascular zone between the cones and the cortical substance are uniformly distended with blood, the cones are usually redder than the cortical substance, and from

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the engorgement of their vessels, and the altered condition of their tubules, they present a series of alternating red and white lines, converging to the apex of the cone, at which point the white distinctly predominate. The pelvis of the kidney is natural.

On microscopic examination with a low power, the congestion of the vessels and the alteration of the tubules become more distinct, the vessels are, for the most part, gorged with blood; the malpighian bodies appear as if injected with red material, or, what is more common, they appear dense and opaque, of an ashen grey colour. In the earliest period of the inflammation, the former condition occurs; in the more advanced period, the latter. Many of the tubules, especially those of the cortical substance, appear darker and denser than natural; sometimes appear as solid bodies, sometimes as tubes, with thickened and somewhat opaque walls. In the groups of tubules passing down towards the cones, and in the cones themselves individual tubules here and there are affected like those of the cortical substance, and contrast strikingly with their neighbours which are unaffected. —*Plate* i., *figs.* 1 *and* 2. On examination with a higher power (300 or 400 diameters), the malpighian bodies appear dense and granular, and where the tubules expand to receive the tuft of vessels, swollen and granular cells may be traced. The tubules are more bulky than natural, their epithelium is swollen, granular, and dense, the cell wall indistinct, and the nucleus frequently imperceptible. On examining a transverse section, this enlargement of the epithelium becomes very apparent, for the cells appear large and granular,

and the lumen is greatly reduced, or altogether lost. In the tubules so altered, a transparent homogeneous material is frequently seen, which fills up the lumen of the tube, and binds together, or, so to speak, cakes into one mass, the epithelium of the tubules, and not unfrequently blood corpuscles are associated with this exuded matter.—*Plate* i., *fig.* 3.

Such are the characters of a typical example of this condition. Numerous varieties are met with—varieties in the *portions* of the tubules affected, for it may be those next the malpighian bodies, or more especially those passing to the cones; in the *amount*, for it may affect the great mass of the organ, or a comparatively small portion; in the *character*, there being a greater or less tendency to rapid desquamation of the epithelium; in the *time* it occupies, coming on slowly or with great rapidity.

In many cases, happily, recovery takes place at this stage of the disease by gradual cessation of the inflammatory action and removal of exuded matter, but in others it goes on and leads to the second stage, which we now proceed to describe.

2*d*, *The stage of fatty transformation.*—In this stage, to which the term "large fatty kidney" is commonly applied, the inflammatory process has passed away, or become very chronic, but its effects remain. The organ is enlarged; its capsule is natural, easily stripped off; its surface is smooth, or slightly depressed here and there. It is pale and fatty in colour, and on its surface stellate vessels are frequently conspicuous. The colour is peculiarly mottled from the mingling of opaque sebaceous-looking fatty portions with the whit-

ish more translucent tissues natural to the organ. On section, the cortical substance is pale, of a yellowish-white colour, and increased in volume, while the cones are pink, and of natural colour and size. The malpighian bodies do not project prominently, as in the first stage. On closer inspection, the sebaceous-looking parts may be seen to correspond to distended convoluted tubules, and not unfrequently lines of this material may be seen running between the small arteries towards the cones.

On microscopic examination with a low power (50 diameters), the tubules are seen in many parts distended with a black untransparent material; this is most marked in the convoluted, but in the straight tubules, here and there individuals or groups may be found affected. The malpighian bodies are enlarged, but not prominent; and, while they may be finely granular, never present the black appearance of the tubules. —*Plate* ii., *figs.* 1 *and* 2. Under a higher power (300 to 400 diameters), the distended tubules are seen to be filled with fatty granules, which, for the most part, are contained within the walls of epithelial cells, which again are embedded in a material which blocks up the tubules. The tubes are, moreover, found to be irregularly distended, in some parts much dilated, in others narrow, or of the natural calibre. In the malpighian bodies, oil globules and fatty cells are frequent, but the capillary tuft is unchanged. The parts in which the fatty condition is revealed by the microscope correspond to those in which the dense sebaceous-looking material is seen by the naked eye.—*Plate* ii., *fig.* 3.

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M^c Farlane, Lith^r Edin^r

3*d*, *The stage of atrophy.*—The organ is diminished in bulk and weight; its capsule is natural, but is less easily torn off than in health; and, on its removal, the surface is found to be very uneven, numerous depressions alternating with elevations. A few stellate vessels are usually seen on its surface. The colour is, as in the second stage, mottled; but there is a greatly smaller proportion of the sebaceous-like untransparent matter than exists in it. On section, the diminution of bulk is found to have been mainly or exclusively at the expense of the cortical substance; that while the cones remain nearly of their natural size, the cortical substance is small and atrophied, and that which intervenes between the cones is greatly diminished. Not uncommonly about the pelvis a deposit of adipose tissue has taken place. In the cortical substance, the malpighian bodies are not prominent, the dense sebaceous-looking matter exists in quantity, but not so abundantly as in the second stage, while the vessels, and especially the arteries, are thicker and more prominent. On microscopic examination under a low power (50 diameters), we at once remark the unusual prominence of these arteries, and thickening of their walls, the relative increase of the fibrous stroma of the organ, and the occurrence of dense dark matter in portions of convoluted tubules; but many of the tubules are shrivelled, atrophied, closed, and the exuded material lies here and there in scattered patches. Many of the malpighian bodies, much reduced in size, are surrounded by a rather thick sheath of fibrous structure. They do not constitute prominent objects, and their size varies remarkably. By higher powers

(300 to 400 diameters), we ascertain the points to which we have referred more distinctly, and make out that the black material contained in the tubules is the same as that seen in the second stage.

How are these different stages related to one another? The first stage is that of inflammation, in which exudation is poured out, and a destruction of the epithelium takes place. This exudation affecting a large number of tubules leads to enlargement of the organ, and also to fatty degeneration of the epithelium, and its absorption or removal leads to ultimate atrophy.

The disease may prove fatal at any stage of its course. Patients not unfrequently die in the first stage; rather more commonly the second proves fatal; but many live till the third stage is well advanced. Recovery happily often takes place after the first stage; complete recovery sometimes occurs, and partial recovery is common, after the second; and it appears that even during the third stage a considerable immunity from unfavourable symptoms may be enjoyed. As to the time required for passing through these different stages I cannot speak positively; it is obvious, that according to the character of the first stage, the duration must vary. I have seen the second stage typically developed within three months of the commencement of the disease, and I have found the third stage well marked within a year of the occurrence of the earliest symptoms.

CHAPTER III.

THE INFLAMMATORY FORM.

Clinical History.

After exposure to cold and wet, or, it may be during or after an attack of scarlatina, erysipelas, or other febrile affection, or without apparent cause, an individual feels some lumbar pain, has frequent calls to micturition, but makes little water at a time, and that of a dark bloody or smoky colour, containing a large amount of albumen, and throwing down a deposit composed mostly of tube-casts. The total quantity of urine is much diminished. The face, the legs, or the scrotum, or all of them together, become œdematous, and the œdema more or less rapidly increases and extends. There is at the same time some degree of febrile disturbance, the breathing may be interfered with, and the patient may complain of headache and drowsiness. On microscopic examination of the urinary sediment, it is found composed mostly of tube-casts, and these present considerable variety of character. Some are composed entirely of granular epithelium, such as is commonly seen in the tubules of the kidney. Others are granular, the outlines of the cells only recognisable on very careful examination. Others contain more or less hyaline material mingled with and connecting together granular cells. And, lastly, some are found in which blood

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CHAPTER II.

THE INFLAMMATORY FORM.

Morbid Anatomy.

THE inflammatory form of Bright's disease is an affection sometimes of short, sometimes of long, duration. When it runs through its whole course, the kidney undergoes a series of changes, which may, for convenience in description, be divided into three stages, each characterised by very distinct anatomical characters, viz.—1st, that of inflammation; 2nd, that of fatty transformation; and 3rd, that of atrophy. We shall now describe these in their order.

1st, *The stage of inflammation.*—The organ is of the natural size, or somewhat larger; its capsule is unaltered, and strips off readily; its surface is smooth, more or less congested, often pink, it is sometimes of a dark purplish colour, sometimes mottled, pale, and purple. On section, the cortical substance is relatively somewhat increased in volume. It is often congested, the malpighian bodies standing out prominently from the surrounding tissue, the congested vessels separated by a varying amount of white somewhat opaque deposit, composed of the altered tubules, to be presently described. The vascular spaces between the cones and the cortical substance are uniformly distended with blood, the cones are usually redder than the cortical substance, and from

of albumen, and throws down a slight deposit of tube-casts, which are mostly hyaline, but the dropsy never wholly disappears, the patient cannot return to work, or, if he does, he has a relapse, and sooner or later his symptoms become aggravated, and he dies with increase of dropsy, or with uraemia, almost constantly with diminution of urine. In such cases we find atrophy, more or less advanced.

In illustration of the Clinical History thus given in outline, I select the following cases :—

Cases Illustrative of the First Stage.

A. Terminating in Recovery.

Case I.—*Acute nephritis ; recovery.*—A. D—, a baker, æt. 30, was admitted to the Royal Infirmary, under the care of Dr Laycock, November 5th, 1859. The patient stated that he had always been healthy until October 18th, when he caught a severe cold. On November 1st he observed that his face and afterwards his body and extremities became swollen. The œdema gradually increased until the date of admission.

He then was intensely œdematous. His urine was scanty, about two ounces in the day, of a pale straw colour, highly albuminous, with a considerable sediment, consisting of granular tube-casts.

Under treatment by dry cupping, diuretics, &c., he soon improved. The urine increased in quantity, the albumen diminished, and the dropsy gradually disappeared. He was dismissed apparently quite well November 25th.

Commentary.—In this case the malady resulted from exposure to cold, and under treatment he completely

recovered within a month of the commencement of his illness. The symptoms, although well marked, were never very severe.

CASE II.—*Inflammation of kidneys following scarlatina; recovery.*—Hugh Bryden, æt. 8, was admitted to the Royal Hospital for Sick Children, under my care, March 2d, 1867.

Soon after admission a scarlatinal eruption appeared. The case was mild, and all went on well for three weeks, but the patient was then seized with symptoms of acute nephritis. He was treated with hot air baths and diuretics, and soon began to improve, although the urine did not return to its normal quantity and quality, continuing to be dark in colour, and to contain albumen. On the 14th of May, however, he became flushed, and began to complain of pain in the abdomen, and to vomit a thin greenish matter, there was no increase of dropsy, but the pulse was rapid, weak, and irregular, and there was slight dulness at the base of the right lung posteriorly. His urine was greatly diminished in quantity, being only two ounces in twenty-four hours, and bloody. By five P.M. on the 16th, he had become comatose, and his breathing was of a stertorous character. He was then cupped over the right loin by the resident physician, Mr Perkins, and an ounce and a half of blood was drawn, with the effect of producing marked and speedy relief. On the 17th, he was much more lively, answered questions, the sickness was relieved, and he passed twenty-two ounces of urine. Ordered five grains of acetate of potash thrice a-day. On the 18th he passed thirty ounces of urine, which contained albumen and granular casts, with a few blood and pus corpuscles. On the 20th the urine was much clearer, casts and blood diminished, albumen about the same as before. On the 21st he was much better, urine greatly improved, the quantity of albumen decreased. On the 22d the urine was almost normal, only a small quantity of albumen being present. The general symptoms improved. On the 24th not a trace of albumen existed in the urine; and on the 1st June the patient had so far recovered, that he was permitted to leave the hospital, at his mother's request.

Commentary.—This case of post-scarlatinal nephritis was at first somewhat mild, although sufficiently distinct; but on the 14th of May, probably after some exposure, suppression of the urine occurred, and I believe that, but for the timely interference of the resident physician, a fatal result would soon have ensued. The after treatment was by means of mild diuretics, and the recovery was complete.

CASE III.—*Inflammation of kidneys following measles; recovery.*—Jane Gibson, æt. 4, was admitted to the Royal Hospital for Sick Children, under my care, May 13th, 1867.

Patient had been admitted into the Hospital on the 28th of April suffering from measles, and was discharged convalescent on the 10th May, which was cold and wet. Almost as soon as she got home she was taken ill with shivering, the skin being at the time very hot. This continued till the 13th, when she was again admitted. For the first few days nothing could be made out farther than that she was more than ordinarily drowsy, but on the 23d, her feet were noticed to be swelled, and she complained of pain in the loins. The urine was diminished in quantity, dark in colour, and contained albumen. A poultice was applied to the back, and she was ordered a diuretic and diaphoretic mixture.

On the 24th of May, she passed only 11 ounces of water. On the 25th, she passed 8 ounces, containing a large quantity of albumen, with granular casts and a little blood. On the 29th, she was much better. Passed 22 ounces of urine with less albumen. On the 30th, the urine was 29 ounces, with a very slight trace of albumen. 12th June.—Case has gone on favourably, and to-day patient was discharged quite well.

Commentary.—In this case, which came on insidiously, and was never very severe, I relied simply upon the fomentation over the loins, and the diuretic and diaphoretic mixture. The result was quite satisfactory.

B. TERMINATING FATALLY.

CASE IV.—*Acute nephritis following erysipelas, fatal on the second day, complicated with pneumonia, pericarditis, &c.*—Mrs B—, æt. 44, was admitted to the Royal Infirmary, under the care of Prof. Bennett, on November 21st, 1865, for eczema. On the 7th of January, when nearly recovered from the eczema, she became affected with erysipelas of the head and face, accompanied by great nervous excitement. On the 12th her urine became albuminous, and she died on the 13th.

Autopsy, twenty-four hours after death.—The scalp was inflamed and thickened, infiltrated with serum. The skull-cap was of natural thickness. The membranes of the brain were congested. There was some serous effusion in the subarachnoid space. The substance of the brain was congested and œdematous. There were slight traces of pericarditis, a little recent lymph coating the right auricle. The lower part of the right lung was in a state of red hepatization. Near the apex there were several masses of old tubercle. The left lung was natural. There were some pale thickened patches on the capsule of Glisson. The substance of the liver was natural. The spleen was natural. There were a few tubercular ulcers in the intestines. The kidneys were of natural size, not congested. The cortical substance had a peculiar homogeneous appearance. The surface was smooth.

On microscopic examination no free exudation was seen in any of the tubules, but the epithelium was in many parts granular and swollen. The malpighian bodies were peculiarly dark, and their epithelium was cloudy and granular. No desquamation of the epithelium at any part could be detected.

Commentary.—The patient evidently died of the results of a blood poison, which affected the lungs, the brain, and the kidneys, and which may probably have resulted from, or perhaps caused, the erysipelas. It presents an example of a very early stage of the disease, the albumen having appeared in the urine only a few hours before death.

CASE V.—*Acute nephritis, complicated with peritonitis, fatal in an early stage; no dropsy.*—H. J—, æt. 17, was admitted to the Royal Infirmary under the care of Dr Sanders, December 12th, 1865.

The patient was a prostitute, and had begun to menstruate about ten days before her death. She had some vomiting and purging, which came on suddenly after a severe fright. She was admitted to the Hospital comatose, but quite free from dropsy. She died thirty-six hours after admission. During that time she passed a small quantity of urine, which contained albumen, and deposited a copious precipitate of granular epithelial tube-casts.

Autopsy.—The body was well nourished; the heart was natural; the lungs were congested, and the bronchi contained fluid. There was general acute peritonitis, the folds of intestine matted together by recent lymph, and coated with pus. The liver was somewhat pale, the outlines of its lobules distinct. There was some fatty degeneration of the peripheræ of the lobules. The spleen was natural. The mucous membrane of the intestines was natural. There was much congestion about the Fallopian tubes and ovaries, and it appeared probable that the peritonitis had resulted from irritation connected therewith. The kidneys were slightly enlarged, in some parts congested, their cortical substance of an ash-gray colour. The epithelium in all the tubules was swollen, cloudy, and granular, undergoing a rapid fatty degeneration. Numerous casts were easily scraped from the surface, but the weight of a covering glass sufficed to break them down into a granular debris.

Commentary.—In this case the renal disease may have been secondary to the peritonitis, or, perhaps they may both have been results of one morbid poison. The death, at all events, occurred in an early stage of the inflammatory form of Bright's disease. In these two cases the fatal result may have been favoured by the renal disease, but was mainly due to the other maladies.

CASE VI.—*Acute nephritis consequent upon scarlatina, fatal in the first stage, with dropsy and uræmia.*—J. R., æt. 20, was admitted to the Royal Infirmary, under my care, on February 21st, 1868.

The patient had enjoyed good health until four weeks before admission, when he had a mild attack of scarlatina. He only kept his bed for a day and a-half, and remained in the house for five days altogether, and then resumed his work. There was very little desquamation. He worked for eight days, when, after exposure to wet, he was again taken ill with sore throat and a general cold. He was then unable to work, and his appetite failed, but he still went about, until he noticed that his feet began to swell.

On admission.—The face was swollen and puffy, but there was little general anasarca. The urine was smoky in colour, of sp. gr. 1012, acid, becoming nearly solid with heat and nitric acid. It contained tube-casts, hyaline, granular, and bloody, and free blood corpuscles. The first sound of the heart was prolonged, and of a blowing character. The precordial dullness was increased. The pulse was 60. The chest was normal anteriorly, posteriorly there was dulness and feeble respiration, particularly towards the base and on the left side. The respirations were 24 in the minute. The tongue was red in the centre, the sides coated with a brown fur. He had considerable thirst and little appetite.

He was ordered to be kept warm, and to take a diuretic mixture. On the 22d he appeared better, passed a good quantity of urine—but that afternoon he exposed himself to cold, getting up and going out of the ward.

In the evening he had a convulsion fit which lasted about four minutes, the dulness over the lungs and the embarrassment of respiration increased. He coughed up a large quantity of watery blood-stained mucus. At 3 A.M. he had another fit. On the following day he was cupped to 6 oz. over the loins, and dry cupped over the back of the chest. The urine was still in good quantity but darker in colour. The œdema of the lungs, however, still increased, and he died on the evening of the 23d.

Autopsy.—There was some degree of general dropsy. The heart was of natural size—contained firm post mortem clots, and in addition, in both ventricles, particularly the right, there were

connected with the valves white granular clots of older date, closely resembling vegetations. The left lung was adherent, the adhesions œdematous. The substance of both lungs was œdematous. The liver, spleen, and gastro-intestinal canal were normal. The kidneys were somewhat enlarged, and considerably congested. The cortical substance was swollen. The tubules contained exudation, and the epithelial cells were granular—in a state of cloudy swelling. They presented, in fact, a typical example of the first stage of the disease as before described. The brain was not examined.

Commentary.—This lad presented no unfavourable symptom on admission, excepting the degree of œdema of the lungs. The increase of dropsy, and the fatal termination, were probably due to his exposing himself to cold on the afternoon of the day before his death; certainly, we must look to some cause acting on the lungs as well as on the kidneys, for the flow of urine was considerable to the last. I do not know whether the peculiar fibrinous clots which had apparently been formed in the heart before death might have had to do with the fatal termination—possibly they might. It is an interesting fact that two brothers of my patient, who had had scarlatina at about the same time as himself, died with symptoms very similar to those which he exhibited. They were under the care of my friend Dr Bryce of Dalkeith.

CASE VII.—*Acute nephritis following pneumonia; death.*—Alexander Jackson, aged 60, a labourer, was admitted to the Royal Infirmary under my care, December 19, 1866.

He had been a healthy man until the 13th, when he had a severe shivering, and from that time was unable to work. On admission he had distinct pleuro-pneumonia of the lower part of the left lung, pulse was 96, and the urine natural. This attack

was going on favourably, although somewhat tardily; but, on the 27th, he had another rigor. His feet began to swell, his urine diminished in quantity, deposited copious lithates, and contained much albumen. The quantity of urine kept low, on the 7th January it amounted to 12 oz., its specific gravity was 1028, it contained much albumen, with blood corpuscles and bloody tube-casts. The dropsy steadily increased. The patient sank, and died on the 9th of January.

Autopsy.—The body was somewhat œdematous, particularly the hands and the lower extremities. The *left lung* was adherent throughout its whole extent. It was œdematous, and its lower two thirds were hepatized. The *right lung* was congested and œdematous. The *liver*, *spleen*, and *gastro-intestinal canal* were natural. The *kidneys* were large and swollen—moderately congested, their capsule stripped off readily. On microscopic examination, the tubules were opaque, their epithelium in a state of cloudy swelling, presenting a typical example of the first stage of inflammatory Bright's disease.

Commentary.—This case afforded an excellent example of a condition which I have repeatedly observed,—viz., the supervention of nephritis on pneumonia—a complication which, in a great majority of cases, proves fatal. My prognosis was most unfavourable from the time that the albuminuria appeared.

Cases Illustrative of the Second Stage.

A. Terminating Fatally.

Case VIII.—*Acute nephritis following upon exposure to cold, dropsy; fatal in second stage.*—B. M—, a servant, æt. 17, unmarried, was admitted to the Royal Infirmary, under the care of Professors Laycock and Bennett, January 20th, 1860.

Previous history.—Had always been healthy until fifteen months before admission, when, after having had her feet wet, she observed them to be swollen. This gradually subsided, but she felt

ill, being subject to occasional palpitation, increased by exertion. On January 6th, a fortnight before admission, her stockings got very wet, but she continued to wear them during the rest of the day. Next morning her face and legs were swollen, and she felt considerable palpitation. As these symptoms did not diminish, she sought admission to the Royal Infirmary.

On admission.—The face and legs were œdematous, and she appeared anæmic. The heart sounds were natural. There was some embarrassment of respiration, and sibilant and snoring rales were heard over the chest. There was some dulness towards the base of the lungs. The digestive and nervous systems were normal. There was considerable tenderness on pressure over the kidneys. The urine was small in quantity, muddy, and deposited a precipitate composed of blood corpuscles and tube-casts, some bloody and some granular. It was highly albuminous. She was treated by diuretics, by dry and wet cupping over the kidneys, by various other plans, without material benefit. The dropsy gradually increased, also the embarrassment of breathing, and although the amount of urine increased, the blood disappeared from it. Casts with fatty epithelium replaced the granular and bloody casts formerly so abundant, and among them not a few hyaline casts were found. Sloughing sores formed on her legs, and she died with symptoms of pyæmia on April 14th.

Autopsy.—Only the kidneys were examined. They were enlarged; the surface was smooth, mottled with yellow, fatty, sebaceous-looking matter; the capsule easily peeled off. On section the cortical substance was found enlarged, dense, opaque, and fatty, and on microscopic examination the tubules of the cortex and some of those of the cones were found distended with exudation and fatty matter. There was no change in the vessels or stroma.

Commentary.—This case lasted for three months and a week, and the second stage of the renal affection had been reached. There was not a trace of atrophy. The whole of the cortical substance of the kidneys was shut off from functional activity, and, unless some means had been successful in removing the exuded material, recovery was almost impossible.

CASE IX.—*Nephritis fatal in second stage.*—E. C—, æt. 48, a washerwoman, was admitted to the Royal Infirmary, under the care of Dr Bennett, March 14th, 1860.

History.—She had always been healthy until October 1859, when she got her feet wet during a menstrual period. She then felt cold and shivering, but continued to work during the day; at night she had distinct rigors, and she observed that the quantity of her urine diminished, and that it was of a dark colour. She continued to work with the aid of stimulants for three weeks, but at the end of that time dropsy of her feet and ankles compelled her to take rest. The swelling steadily increased. In the beginning of February she felt some difficulty of breathing, and complained of a cough. All these symptoms increasing, she sought admission to the Infirmary.

On admission.—There was general anasarca; the abdomen was much distended, measuring forty-four inches in circumference. There was no pain on pressure over the kidneys. The urine was in small quantity, smoky, of sp. gr. 1010, very albuminous, and threw down a thick whitish sediment, composed of granular and fatty tube-casts, with some blood corpuscles. The tongue was furred, the appetite bad, the bowels constipated. There was much dyspnœa, considerable consolidation of the left lung posteriorly. The cough was frequent, and accompanied by a tenacious purulent expectoration. The heart was natural.

After admission.—Under powerful diuretics the quantity of urine rose, but again gradually subsided, notwithstanding the continuance of the drugs. Bitartrate of potash seemed to be the most useful, but appeared to increase the quantity of blood discharged by the kidneys. The dropsy never materially diminished, and the dyspnœa was constantly increasing. She died exhausted, on April 7.

Autopsy.—There was general anasarca, and great ascites. There was also effusion into the pleuræ, and a considerable amount of mucus in the bronchi. The heart was natural. The liver was congested, weighed three pounds. The spleen was small and dense, weighed two ounces. The intestines were healthy. The kidneys were enlarged, pale, and mottled; they weighed together fifteen and a half ounces. The capsule was easily stripped off. The cortical substance was swollen, opaque, and fatty. The

tubules were found, on microscopic examination, distended with exudation, the epithelium to a great extent fatty.

Commentary.—This was a less acute case than the last. It proved fatal in the second stage, and had existed for six months.

CASE X.—*Nephritis following upon cold, fatal in second stage; uræmia, dropsy, apoplexy.*—J. P—, æt. 40, was admitted to the Infirmary, under the care of Dr Laycock, February 15th, 1865. She was a married woman, of temperate habits. About the middle of November 1864, she was exposed to cold and wet. She then became affected with lumbar pain, swelling of the face, afterwards of the legs, and ultimately of the whole body. The urine was high-coloured, and diminished in quantity.

On admission.—She had symptoms of bronchitis. The whole body was œdematous. Her urine contained much albumen, and a large number of granular tube-casts. She did not improve under treatment, but gradually became worse until the middle of April, when she was seized with convulsions, afterwards became comatose, and died April 16th.

Autopsy.—The body was very œdematous. The peritoneum was distended, and there was a considerable amount of fluid in the pleuræ and pericardium. The heart was natural. The lungs were congested and œdematous. The liver was small, congested, and fatty. The spleen was natural. The kidneys were enlarged, and presented the characters of a typical example of the large fatty kidney, the second stage of the inflammatory form of Bright's disease. There was some serous effusion into the subarachnoid space, and in the optic thalami there were recent clots.

Commentary.—This case was an example of the inflammatory form, terminating during the second stage, five months after the commencement of the malady. The death did not result directly from the renal disease, but from the apoplexy.

B. Terminating in Partial or Complete Recovery.

Case XI.—*Acute nephritis; partial recovery during the second stage.*—R. P—, æt. 26, a maltster, had been healthy until January 1865. He had been originally a baker by trade, but had four years before become a maltster; in both businesses was much exposed to vicissitudes of heat and cold.

About the end of January 1865, he noticed his feet swelling. The œdema increased rather rapidly. He had frequent calls to micturition, but he made little water at a time, and that dark and bloody. He was admitted to the Infirmary on March 3d. Under diuretics the urine increased in quantity, and he somewhat improved, but the dropsy again increased and the urine diminished. When he came under my observation, on April 27th, the following was his condition:—The face was pale and pasty. There was great dropsy of the legs and feet, scrotum, penis, and flanks, and considerable ascites. The urine was pale, but smoky, of sp. gr. 1020, highly albuminous, and deposited a precipitate containing numerous casts, hyaline and fatty; the quantity was forty ounces. There was a good deal of bronchitis. Hot-air baths and acetate of potash had been tried for some time. He was ordered, April 28, a dessert-spoonful of infusion of digitalis three times a day, and on the following day a drachm of electuary of bitartate of potash was added. These did not induce diuresis, indeed, the urine rather diminished in amount and the dropsy increased. On May 3d, he was ordered to inhale forty minims of oil of juniper twice a day. The quantity of urine at once increased, and the dropsy began to diminish. The quantity of water steadily increased, rising from forty-six ounces on May 1st, to eighty-four on the 12th, and afterwards to more than one hundred ounces. On May 11th, a few pricks were made with a needle in the left leg to relieve the obstinate dropsy, and a considerable amount of water drained away. On the 24th of May the dropsy had disappeared.

From this time he pretty steadily improved. The dropsy was quite gone. The urine in good quantity, with a natural amount of urea and salts, and the albumen diminished. The fatty were replaced by hyaline casts. He was dismissed on August 8th, quite well, excepting that his urine was albuminous.

In October he was exposed to cold and damp in his situation as light porter, and in consequence had a reaccession of his disease. The quantity of urine was reduced to twenty ounces. It was very albuminous, with tube-casts containing fatty cells. The dropsy was very considerable. Under diuretics he again steadily improved, and was again dismissed on December 15th. The albumen continued at date of dismissal. A few months ago he came to the hospital, said he was quite well, and regularly at work, but his urine still contained some albumen.

Commentary.—The case was a typical one of acute nephritis, passing through the second stage of fatty degeneration, and becoming chronic. The value of diuretics was very apparent, and particularly of the oil of juniper, as first recommended by Sir James Y. Simpson. A distinct increase of the flow of urine followed its administration, and from my experience of it in this and other cases, I think it is a remedy which should be tried in all obstinate cases of dropsy.

Cases Illustrative of the Third Stage.

CASE XII.—*Nephritis fatal in third stage; general dropsy, &c.*—E. R—, a female, æt. 62, was admitted to the Royal Infirmary, under the care of Dr Haldane, in October 1865. She was then extremely anasarcous, passed small quantities of albuminous urine. Notwithstanding the use of powerful diuretics, the amount of urine continued low, the dropsy became more intense, and she died October 13th. She stated that she had been healthy until eight months before, and had then become dropsical.

Autopsy.—The body was very œdematous; the abdomen was distended with clear serum. The pleuræ and pericardium also contained much fluid. The lungs were compressed. In both apices there were traces of old tubercle. The heart was enlarged; the left ventricle was much hypertrophied; the valves were com-

petent; the aorta was atheromatous, in part calcareous. The liver weighed one pound twelve ounces, its substance was congested. The spleen was small. The kidneys weighed together nine ounces. Their surface was granular. On section their cortical substance was found to be infiltrated with fatty sebaceous-looking matter, and in a state of commencing atrophy. On microscopic examination many of the tubules were found to be fatty and some in a comparatively natural state. The intestines were very œdematous.

Commentary.—This case had lasted about eight months, and the third stage of the disease had been reached. The hypertrophy of the left side of the heart had resulted partly from the atheroma of the vessels, and partly from the renal affection. The death was due mainly to the intense dropsy, which was in part referable to the disease of the circulatory system, in part to the renal affection.

CASE XIII.—*Nephritis, fatal in third stage; general dropsy, &c.*—E. T—, æt. 30, was admitted to the Royal Infirmary, under the care of Dr Haldane, April 3rd, 1865. In summer 1864, she had caught cold, in consequence of which her urine diminished in quantity, and became high-coloured. She then also had dropsy. She never fully recovered. While under treatment she had dropsy to a considerable extent, and sank exhausted.

Autopsy.—The body was œdematous. The pleuræ, pericardium, and peritoneum contained clear serum. The heart was enlarged and dilated. The left side was much hypertrophied. The right side was dilated. The valves were free from disease. The lungs were partially collapsed from the pressure of the dropsical fluid, but their margins were emphysematous. The liver weighed two pounds six ounces. Its capsule was thickened. The spleen was natural. The kidneys weighed together seven ounces. Their surface was granular; the capsule peeled off readily. On section the cortical substance was found pale, fatty, and partially atrophied. Many of the tubules contained fatty matter within the epithelial cells.

Commentary.—The case afforded an excellent example of this form of disease in an advanced stage. The organs were considerably reduced in size. The marked hypertrophy of the heart appeared to depend mainly upon the renal affection. The disease had lasted fully a year.

CASE XIV.—*Nephritis, repeated acute attacks, fatal in the third stage, with uræmia and general dropsy.*—P. M.—, æt. about 30, had been for upwards of a year under Dr Haldane's observation in the Royal Infirmary. Early in 1864 he had acute nephritis, from which he gradually recovered. He then had repeated acute exacerbations, and ultimately came into the hospital with severe general dropsy and diminished flow of urine. The urine was very albuminous, and contained many fatty and hyaline casts. The quantity of urine did not increase, uræmic convulsions came on, and death ensued on January 5th, 1865.

Autopsy.—There was general anasarca, and dropsy of the serous cavities. The lungs were œdematous, contained some carbonaceous deposit. The bronchi were congested. The heart was enlarged. The left ventricle was much hypertrophied, the right dilated. There was no valvular disease. Both the liver and spleen were congested. The intestines were congested, otherwise natural. The kidneys were of about the natural size, their surface was granular, mottled, and opaque. The capsule was somewhat adherent. The cortical substance was relatively somewhat diminished in size. On microscopic examination many of the tubules were found full of dark fatty matter, while some had natural epithelium. The brain was somewhat œdematous, the arteries at its base atheromatous.

Commentary.—In this case the disease had lasted fully a year, and the third stage had been fully established. The hypertrophy of the heart was in part due to the renal affection, and in part to the disease of the arteries.

PLATE III.

CHAPTER IV.

THE INFLAMMATORY FORM.

Nature of the Symptoms.

THE symptoms which deserve special notice are those connected with the urine, the dropsy, and the affections of the nervous system.

I. *The urine* is diminished in quantity; commonly dark, smoky, or bloody in colour; albuminous; and contains tube-casts. The diminution of its quantity depends upon the congestion, and the occlusion of the tubules. In the early stage of the disease the former, in the more advanced stage the latter cause especially prevails. The albumen and the blood escape from the renal capillaries in consequence of the inflammatory action, just as they do from those of the lungs in cases of pneumonia, and thus we account for their occurrence in the inflammatory stage. It has been proved by Dr Robinson of London (formerly of Newcastle), and I have satisfied myself by experiment of the correctness of his statements, that tying a renal vein leads to extreme congestion of the corresponding kidney, and the escape of albumen, fibrine, and blood into the tubules; and it is certain that inflammatory congestion leads to the same results. But why do we find albumen still present in the advanced stages when inflammation has apparently ceased? It may arise from various circumstances. In some cases, a

certain degree of inflammatory action may continue throughout the whole course of the disease. If a considerable portion of the kidney is shut off from action, that which continues to act may well be supposed to be subjected to an amount of blood pressure sufficient to lead to the transudation of albumen along with the water of the blood. It seems very reasonable to suppose, although I do not know that it is capable of demonstration, that when the basement membrane has been denuded of its epithelium, the albumen of the blood may transude through the capillary walls along with the water, and as many tubules are in this condition in the more advanced stages of the disease, we may find in this also a partial explanation. Lastly, it is possible that the albumen of the blood may be so altered in its diffusibility that it may transude through the capillary walls.

The *Tube-casts* are composed of coagulated fibrine, with altered renal epithelium, and not unfrequently blood corpuscles. Their characters are very various, but the following are the chief varieties :—

1. Bloody casts, in which, along with fibrine, and, it may be, epithelium, blood corpuscles are present.—(*Plate* iii., *fig.* 1.) The presence of such casts implies the rupture of small vessels.

2. Granular epithelial casts, in which there are numerous epithelial cells along with the fibrine. The cells are for the most part granular and opaque, presenting the characters of the unhealthy epithelium, as described under the head of morbid anatomy.—(*Plate* iii., *fig.* 2.)

3. Hyaline casts, which are simply fibrinous. They

vary much in size, and it appears that they are sometimes formed in the lumen of tubes still lined by epithelium, sometimes in tubules whose epithelium had been removed.—(*Plate* iii., *fig.* 3.) It is very unfortunate that the term "waxy cast" has been applied to this form, for it has no connection whatever with the waxy degeneration.

Such are the forms of casts met with in the first stage; in the second and third stages the blood casts do not as a rule occur, and the epithelial are less numerous, the prevalent forms being the fatty and the hyaline.

4. Fatty casts are those in which the epithelial cells contain fatty granules; in some cases the cells thus degenerated are so numerous, that the whole mass appears composed of fatty matter; in other cases they are mingled with more or less hyaline material. In many instances, particularly when the disease is well advanced, casts occur composed almost entirely of fibrine, with only a fatty cell, or a group of fat granules representing a cell, here and there.—(*Plate* iii., *fig.* 4.) The hyaline casts in the advanced stages are often of large size.

In the first stage, and sometimes in the more advanced conditions, the urea is diminished, but I have found, in several cases in the second and third stages, that its quantity was natural. The chlorides are diminished in the early stage, as are also the sulphates, phosphates, and uric acid. In chronic states these conditions are less marked. For further information upon these points, I would refer the reader to Dr Dickinson's work.

II. *Dropsy* is an early and prominent symptom, but

is generally preceded by abnormality of the urine. It is in most cases general, but is usually first observed in the feet and ankles, the eye-lids or the scrotum. The anasarca is often accompanied by effusion into serous cavities, but such effusions do not occur without general œdema. In chemical composition it is found to be a watery fluid of low specific gravity, with little albumen, and a large proportion of salts. C. Schmidt* has shown that the amount of albumen is in so far determined by the system of capillaries through which the transudation occurs. He found it most abundant in pleural effusions, less so in peritoneal, still less in the cranial serous cavities, and least in the subcutaneous cellular tissue. Lehmann has shown that the slower the circulation in the capillaries is, the richer in albumen is the dropsical effusion. The poorer the blood is in albumen, the less albuminous is the dropsical fluid. In long standing dropsies the albumen becomes relatively greater from re-absorption of the water and salts. If the blood be rich in urea, that substance may also be found in quantity in the dropsical fluid. The dropsy may, in the first stage, be ascribed to deficiency of albumen in the blood serum, a condition which appears to arise very early in the disease; but it appears doubtful whether this may be regarded as the sole explanation, as dropsy is often the first symptom observed, and is frequently more marked in the commencement than it is in more advanced stages, when the blood has been deteriorated by a long continued drain of albumen. It may, again,

* See Uhle und Wagner's Handbuch der Allgemeinen Pathologie, Dritte Auflage, s. 249. Also, Lehmann's Physiological Chemistry, Cavendish Society's Translation, Vol. ii., p. 318.

be referred to non-elimination of water by the kidneys, and a consequently increased pressure of the blood on the walls of the systemic capillaries and veins. The greater the severity of the renal disease, and the diminution of the urine, the more acute and copious is the dropsy; and as the renal disease subsides, and the water is again discharged by its natural channels, the dropsy disappears.

But there are cases, particularly among those following scarlet fever, in which the dropsy appears simultaneously with, or even before, the arrest of the action of the kidneys. In these it has been suggested that it results from paralysis of the vessels of the skin and subcutaneous tissue, a result of the cold which leads to the renal affection, but it appears more probable that in truth the renal affection has been first established, although its direct symptoms are not yet manifest. A consideration opposed to this explanation is, that we find, as Dr Christison pointed out, that an inordinate flow of urine sometimes accompanies a dropsical state of the cellular tissues. He remarks that this is most common in the middle stage of the primary disease. I have seen it in some cases, but in most, if not in all of them, the polyuria depended upon waxy degeneration of the vessels.

As the disease advances, the impoverished condition of the blood favours the occurrence or hinders the re-absorption of dropsical effusion, for diminution of albumen and increase of water in the blood constantly produces such effects. It must also be borne in mind that cardiac or other disease may contribute to the production of this symptom.

III. The *symptoms connected with the nervous system* are often very important, and have been admirably described by Frerichs. They may be acute or chronic, suddenly appearing with marked intensity, the patient rapidly becoming comatose, unexpectedly convulsed, or both. On the other hand, they may come on insidiously, the patient growing drowsy, the drowsiness passing into torpor, and that into coma, from which he never awakes. Many views as to the causes of these symptoms have been advanced and successively disproved, and at present it may be said that they are still unexplained.

Osborne* sought to refer them to arachnitis, but this view was definitely set aside by the result of post-mortem examinations. Dr Owen Rees † observing that the occurrence and intensity of uræmic symptoms do not always correspond to the quantity of urine, and that the blood may be loaded with urea and yet no such symptom occur, conceived that a certain thinness and watery state of the blood was an essential condition for its production. But as uræmia occurs in many cases where the blood is not watery, and watery blood is not always attended by uræmia, this view cannot be accepted.

Most authors ascribe it to the retention in the blood of some excrementitious material. Urea was the first substance blamed, for Prevost and Dumas‡ had proved its presence in the blood of animals whose kidneys had been extirpated, and Christison and others recognised it in that of uræmic patients. But Bauquelin

* On the Nature and Treatment of Dropsical Diseases, 1837.

† On the Nature and Treatment of Diseases of the Kidney connected with Albuminous Urine, by Owen Rees, London, 1850. ‡ Quoted by Frerichs, op. cit.

and Segalas* injected this substance into the veins of dogs and rabbits without leading to any result beyond an increased secretion of urine. As, on the other hand, they found that the injection of pure urine was fatal, they concluded that all the elements of the urine together constituted the poison. Bichat and others, however, injected filtered urine without injury. Uric acid, urate of soda, and ammonia were injected with like results.

Frerichs† advanced the theory that the symptoms depend on decomposition of urea into carbonate of ammonia and water, which decomposition he ascribed to the action of a ferment in the blood. Urea might accumulate for long in the blood of patients affected with Bright's disease, but would lead to no injurious influence unless a ferment were introduced, and then the symptoms would be rapidly developed. He sought to prove this view by showing that injection of carbonate of ammonia leads to such symptoms, and that carbonate of ammonia is present in the blood of all uræmic patients. These results were, in all points, confirmed by Dr Petroff of Dorpat, who, in an elaborate memoir, supports Frerichs' view.‡ Dr Treitz§ supposes that carbonate of ammonia is the poison, but that it is not formed, as Frerichs supposed, in the blood, but in the alimentary canal; for, he says, when the kidneys do not act, the urea is eliminated by the bowel, there decomposed, and it may again be absorbed into the blood and produce uræmia.

Oppler‖ has found, however, that the symptoms

* Magendie Journ. de Physiologie, Vol. ii., p. 354. † Op. cit., p. 108, et seq.
‡ Virchow's Archiv. Bd. xxv., s. 91. § Prager Vierteljahrschrift, 1859.
‖ Virchow's Archiv., 1861, Vol. xxi., p. 260.

resulting from carbonate of ammonia are by no means identical with those seen in uræmia, and able chemists have failed to discover the salt of ammonia in the blood of the uræmic. Oppler found that there is a retention of the products of muscle waste in cases of Bright's disease, and conceives that there may be a similar retention of the products of nerve waste, and to the deleterious influence of this substance he would incline to ascribe the symptoms.

The experiments of Oppler* and Zalesky† make it appear that urea is formed by the kidneys from nitrogenous materials in the blood, a fact which, if it be confirmed by other observers, will afford further evidence against the earlier theories. Perls and Schottin have confirmed these views.

Since the theory of Frerichs was set aside, no very definite one has been advanced, excepting that of Dr Oppler. One of the most recent writers, Dr Rommelære,‡ of Brussels, conceives that the nervous symptoms are not to be ascribed to one cause, but to many causes combined; for, he remarks, when the functions of the kidney have been interrupted, not only does the waste azotized matter cease to be eliminated, but water accumulates in the system, causing impoverishment of the blood and increased tension of the blood vessels. To the combined action of all of these the nervous symptoms are referable.

Notwithstanding the extraordinary amount of attention which has been given to this subject, we must admit that further observation and experiment are still required for its satisfactory elucidation.

* Loc. cit. † Untersuchungen ueber den Uræmischen Process, Tubingen, 1865.

‡ De la Pathologie des Symptomes Uræmiques, Bruxelles, 1867. Quoted by Dr Bennett, Principles and Practice of Medicine, 5th edition, 1868.

CHAPTER V.

THE INFLAMMATORY FORM.

Complications.

In considering the complications of any particular malady, we should distinguish the following varieties :—

1*st*, Such as result from the malady, Consequent complications.

2*d*, Such as cause the malady, Causal complications.

3*rd*, Such as owe their origin to the same cause as leads to the malady, Concomitant complications; and we might add, as a fourth class, diseases which accidently co-exist with the malady, Casual complications.

The complications of inflammatory Bright's disease are numerous and important, and afford illustrations of all the classes above referred to. We shall consider them in their order.

A. Consequent Complications.

1*st*, *Hypertrophy of the Heart.*—This has long been recognised as a complication of renal disease. I have found it present in 57 per cent of a series of cases of nephritis, fifty-one in number, that I examined in the Royal Infirmary, but of these 17 per cent were affected with other changes capable of accounting for the lesion, still, in 40 per cent of all the cases it

was present, and was apparently solely referable to the disease of the kidney. Further, on examining its relationship to the different stages, I found it specially related to the more advanced, for while in the cases fatal in the first stage it occurred only in 12 per cent, of those fatal in the second stage it was present in 38 per cent, and of those in the third in 100 per cent. Thus it presents, in an eminent degree, the important characteristic of a consequent complication, viz., that it increases in frequency as the disease advances. It certainly is curious that all the cases in the third stage should have presented this complication, and without any cause except the renal malady; but it shows, on the one hand, how unfailingly long standing disease in the one organ leads to an affection of the other, and, on the other hand, how patients who have valvular disease of the heart and inflammatory Bright's disease together do not live to reach the third stage of the latter affection.

The cause of this hypertrophy is doubtless the impurity of the blood. The blood is impure, because the natural eliminant action of the kidneys is diminished; and we know that an impure blood is circulated with greater difficulty than the healthy fluid. In order to overcome this difficulty, the heart enlarges, in accordance with the law which we find prevailing everywhere in the body, that increased function leads to increased growth.

2*d*, *Affections of the lungs and bronchi.*—Two pulmonary diseases occur not unfrequently in combination with this renal disease, viz., congestion and œdema of the lungs and bronchi, and pneumonia.

a. Congestion and œdema of the lungs and bronchi.—This is a frequent complication, and often is the immediate cause of death. It may be acute, developing with great rapidity, or chronic, slowly and insidiously increasing. In my series of cases of the inflammatory form, I found it present in 64 per cent. Frerichs, on the other hand, found it only in 25 per cent. Other writers have regarded bronchitis as a frequent complication, but I have found a truly inflammatory condition of the bronchi exceedingly rare. That mucus or watery fluid is frequently present in large quantity is certainly true, and the existence of this fluid, of course, produces most of the symptoms and physical signs of bronchitis; but this appears to be connected with œdema rather than inflammation, for I have found generally, when the mucus had been washed off by a stream of water, that a healthy or simply congested condition of the mucus membrane existed. It was specially frequent in the cases which proved fatal in the first and third stages of the disease, occurring in 75 per cent of the former, and 85 per cent of the latter. It is evidently a local manifestation of the general dropsy.

b. Pneumonia.—This complication was present in 21 per cent of my cases, was most common in the second stage, and about equally frequent in the first and third stages. This circumstance renders it doubtful, I think, whether it can be regarded as a consequent complication of the class to which cardiac hypertrophy belongs, viz., those depending upon abnormalities of the blood, constantly increasing as the disease advances. At the same time, it should be

borne in mind that this may not be the only class of consequent complications. For it is quite conceivable that as symptoms manifested in the early stage of a disease sometimes disappear as the malady progresses, so complications which are truly consequences may occur in the early stages and be absent in the more advanced. It may be that the system comes afterwards to tolerate irritating materials, which, when first introduced or retained in excess, produced marked effects. We may thus conceive that pneumonia prevails more in the early stages than in the most advanced, and is yet a consequent complication. In many cases, again, the pneumonia is a causal complication, as I shall show further on. In others, again, it is a concomitant, as when both result from a blood poison, *e.g.*, that of pyæmia. In other instances, again, it has seemed to me that the two diseases were merely casually associated. From these considerations, it appears that the number of cases in which pneumonia is a consequent complication are much fewer than my tables would at first sight indicate. Competent observers, however, have conceived that a very marked connection exists between the two diseases, that pneumonia is a common cause of death in nephritis, and directly results from the renal malady.

3*d*, *Inflammation of serous membranes.* — These affections have been commonly regarded as among the most serious and fatal complications of Bright's disease. Sir Thomas Watson says,* "Intercurrent acute inflammation is not an uncommon cause of the patient's death. The pleura appears to be much more

* Watson's Practice of Physic, 4th edition, Vol. ii., p. 682.

often affected in this way than either the peritoneum or the pericardium." Frerichs found pleurisy in 12 per cent, peritonitis in 11 per cent, and pericarditis in 4 per cent of his cases. Rosenstein, again, found pleurisy in 16 per cent, peritonitis in 8 per cent, and percarditis in 7 per cent. Among my cases, I found about 14 per cent of pleurisy, 7 per cent of pericarditis, and no case of peritonitis. My results thus in so far confirm those of the German observers, and the remarks of Sir Thomas Watson; but it is curious to find that it is specially associated with the earlier stage of the disease, for all the cases of pericarditis were with the first stage, and while 7 per cent of those of pleurisy were in that, only 3 per cent each occurred in the second and third respectively. Thus the most important characteristic of one class of the consequent complications is wanting in the case of the serous inflammations.

4th, Derangements of the alimentary tract.—Changes similar to those met with in the kidney not unfrequently occur in the tubules of the stomach, but in not a few cases, on post-mortem examination, the stomach is found little changed. Dr Fenwick* states that, in the majority of those dying from Bright's disease, we find evidence of gastritis on post-mortem examination. The mucus membrane is more vascular, the tubes are readily separated, but are distended with a confused mass of cells and granular material, the basement membrane is sometimes thickened, sometimes normal. Of ten cases of this form of Bright's disease, in which Dr Wilson Fox† carefully examined

* The Morbid States of the Stomach and Duodenum, by Samuel Fenwick, M.D., 1868, p. 177.

† Medico-Chirurgical Transactions, Vol. xli., p. 361.

the stomach, eight had an exactly corresponding condition of the gastric glands, and in two there was in addition thickening of the intertubular tissue. Dr Fenwick has shown * that tubular gastritis is almost invariably present in persons who die of scarlet fever, and that the application of cold to the surface often leads to the same result. The gastric affection may, therefore, in many cases, be regarded as a concomitant complication of the renal. But any one who has carefully watched cases of the kind, must have observed that it arises after, and as a consequence of, the kidney disease, probably as a result of irritation by the excrementitious matters retained in the blood.

During life the digestive organs are much affected. Patients, in all stages of the disease, often complain of nausea, loss of appetite, and vomiting, particularly in the morning. Sometimes, in severe cases, the vomited matter has an ammoniacal odour. These derangements are often little amenable to treatment.

5th, Diseases of the brain.—The affections of this organ which have been most commonly recognised as complications, are sanguineous and the so-called serous apoplexies. But I confine my attention to the former class, having found, like most other recent pathologists, that the anatomical conditions which used to be regarded as characteristic of serous apoplexy are commonly met with in diseases accompanied with no apoplectic symptoms, and are not always to be found in cases whose clinical history would have led former observers to expect them.

Sanguineous apoplexy occurs as a fatal termination

* Op. Cit. p. 92, et seq.

in a certain proportion of the cases of this form of Bright's disease. It occurs, moreover, with increasing frequency as the disease advances; for while among my cases fatal in the first stage it occurred in none, it was met with in 7 per cent of those fatal in the second, and in 14 per cent of those fatal in the third. Doubtless, the embarrassment of the circulation, the defective nutrition of the vessels, and the hypertrophy of the heart, contribute to the frequency of the complication.

6*th*, *Morbid conditions of the blood.*—These changes are well entitled to be regarded as consequent complications, but it must be acknowledged that little or nothing of importance has been added to the observations which Dr Christison recorded in 1829 and 1839.* He showed that in the early stage the blood is characterised by the low density of its serum, the deficiency of albumen, the frequent presence of urea, the frequent increase of fibrine, and by the proportion of hæmatosin being unaffected. The most remarkable change is the decrease of density, falling as it does from the normal 1030 to 1022 or even 1019, and the solid contents being reduced from 100 or 102 in a thousand to 68, 64, or even 61. These changes occur only when there is abundant discharge of albumen with the urine.

In the advanced stages the proportion of hæmatosin in the blood is invariably and greatly reduced. No other morbid change is constantly present, but the solids of the serum are often deficient, sometimes

* Edinburgh Medical and Surgical Journal, October 1829, and Granular Degeneration of the Kidney, p. 59, 1839.

again they are in excess; and not unfrequently the serum contains urea.

B. Causal Complications.

The complications belonging to this class are so important as to deserve separate consideration. They are discussed in the next chapter along with the other causes. One supposed cause only may be mentioned here, as it has been deemed important by some observers.

Tubercle of the lungs.—This affection of the lungs has been regarded by some as a cause, but is in truth not specially related to this form of Bright's disease. It occurred only in 7 per cent of my cases. It was present in 12 per cent of the cases which proved fatal in the first stage, in 15 per cent of those fatal in the second, in none of those fatal in the third. But while the purely inflammatory form bears no relationship to tubercle, in the combined waxy and inflammatory it is a very frequent complication, indeed, in 52 per cent of my cases tuberculosis of the lungs existed. To this we shall refer further on.

C. Concomitant Complications.

The complications belonging to this class are not in this disease of much importance. Only those of the liver and spleen seem to me to be worthy of notice.

Affections of the liver.—It has been long known that the diseases of the liver and kidneys frequently correspond, and there are two conditions which I have found co-existing with this form of Bright's disease, viz., fatty degeneration, which occurred in 25 per

cent of my cases, and cirrhosis, which occurred in 14 per cent. The former condition is frequently associated with nephritis in cases of blood poisoning, the secreting cells presenting identical changes in the two organs.

Affections of the spleen.—In cases like those just mentioned, the spleen is not unfrequently found enlarged and pulpy.

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CHAPTER VI.

THE INFLAMMATORY FORM.

Causes.

THE most common exciting cause of inflammatory Bright's disease, in the adult, is *exposure to cold and wet.* This induces its effect in many cases very speedily, and its action is greatly favoured by the existence of an exhausted state of the system. Among bakers, and other workmen whose employment renders them liable to sudden vicissitudes of temperature, the disease is specially common.

As to the mode of action of cold in inducing renal disease, various theories have been advanced. Some have supposed that as the action of the skin and kidneys in discharging water are complementary one to another, a sudden arrest of either must greatly embarrass the other, a suppression of cutaneous excretion might thus suffice to inflame the kidneys. But, as Frerichs justly remarks, this explanation is unsatisfactory, seeing that when much fluid is taken into the system the kidneys may be made to excrete twice the natural amount of water, and no inflammation result. Another view is that which Dr Johnson* so ably supports, that the defective action of the skin causes certain deleterious matters to accumulate in the blood,

* Dr George Johnson on Diseases of the Kidneys, London 1852.

and that thus the burden of their elimination, a burden which proves necessarily injurious, is thrown upon the kidneys. If it be true that the skin secretes considerable quantities of urea, and that the sweat contains other elements which make it appear closely allied to urine, it may well be believed that a sudden suppression of its action may lead to irritation of the kidney; at the same time, it is remarkable that well-marked renal symptoms may appear almost immediately after exposure to cold. Another view would refer it to the reflex influence of the nervous system; explaining it as we are accustomed to explain many cases of pneumonia, pleurisy, gastritis, catarrh of the intestine, and other diseases. It is true that we cannot tell why the kidneys should be specially affected rather than other organs, still, it can scarcely be doubted that the disease is sometimes produced in this way. Probably in some cases the true explanation is to be found in the second, in some in the last-mentioned theory.

Another great cause of this malady is the presence of *morbid poisons in the blood.* The morbid poisons which are specially active in this way are those of scarlatina, diphtheria, erysipelas, measles, pyæmia, typhus, cholera, ague, rheumatism, and that which leads to acute atrophy of the liver. The scarlatinal poison is, of all these, the most important, because the most frequent originator of the malady. In children, indeed, it is by far the commonest cause of the disease. The nephritis may occur at any time, from the commencement of the fever until the desquamation has been completed. The most common period for its

commencement is from the end of the first week to the fourth; but the danger cannot be said to be over before the end of the second month. In a very large proportion of cases albumen occurs in the urine at some stage of the fever, but the actual nephritis generally results from exposure to cold, and is therefore specially apt to occur in mild cases, and during convalescence.

In pyæmia, I have found the kidneys very constantly affected, but not in an advanced stage, death generally occurring before there was time for all the symptoms to be manifested.

After erysipelas and measles, I have repeatedly observed nephritis arise.

The co-existence of renal disease with acute atrophy of the liver I shall consider in a supplementary chapter at the end of this work.

In all of these cases the inflammation appears to result from the irritation of the kidneys by the morbid poison which causes the primary disease, that poison irritating the kidneys perhaps during the process of elimination.

Occasionally, too, we find nephritis resulting from the presence of irritating substances introduced into the body from without, as cantharides, copaiba and cubebs, oil of turpentine, and, some would add, alcoholic drinks. Bouillaud* carefully investigated the effects of cantharides, and found that almost constantly after the application of large blisters to the skin albumen appears in the urine; and in the dead body, along with a general congestion of the urinary tract, he found

* Archives Générales de Médecine, 4me Série, Tome. xvii., p. 99.

the kidneys much congested, with numerous little extravasations throughout their cortical substance.

Reinhardt* relates two cases which were due to the excessive use of balsam of copaiba and cubebs. Both were in weakly individuals, and one proved fatal.

Similar observations have been made as to the action of oil of turpentine.

With regard to the abuse of alcoholic liquors, it appears that much as such a habit may predispose to renal inflammation, there is no evidence to show that it is capable of directly exciting it.

Another important cause of nephritis is internal inflammation, particularly of the lungs. This cause seems well worthy of attention, for I have found that in a considerable number of the fatal cases of pneumonia which I have examined in the Infirmary, during the past four years, a greater or less degree of inflammation of the kidneys existed, and the urine was in many cases albuminous. I have recorded a very well-marked case of this kind in Chapter IV., a case in which the occurrence of the renal complication was observed from its commencement, and carefully watched throughout its course.

In diabetes mellitus it is well known that nephritis sometimes occurs as a secondary disease. I have, in two cases, seen it come on with such severity as to lead to a rapidly fatal result.

Pregnancy sometimes occasions this form of renal disease. Dr Lever† pointed out, more than twenty

* Annalen des Charité Krankenhauses I Jahrgang, 4tes Heft, quoted by Frerichs, op. cit., s. 149.

† Guy's Hospital Reports, 1843.

years ago, that puerperal convulsions are commonly associated with albuminuria, and it has since been proved that while these convulsions are common in women who have become pregnant while labouring under Bright's disease, that disease is much more commonly found to come on during pregnancy, as a result of it. It frequently appears in successive pregnancies, disappearing completely during the intervals, but in such cases it usually at length becomes chronic and permanent. It may arise at almost any period of pregnancy, but is very rare before the third or fourth month, more common during the later months, sometimes coming on during labour, and after delivery. A close connection, doubtless, exists between the renal affections and puerperal convulsions; but, on the one hand, it is certain that albuminuria may exist during pregnancy, and yet be unattended by convulsions; and, on the other, that the convulsions may precede the albuminuria, or even sometimes occur independently of any renal symptoms. We are indebted to Dr Braxton Hicks* for showing that the convulsions sometimes precede the albuminuria, a fact which he has illustrated by several interesting cases.

As to the mode of production of the disease, Dr Lever suggested that it depends upon pressure of the gravid uterus on the renal vessels. This view has been accepted by many, but it is disproved by the facts that the veins are often subjected to much greater pressure in cases of ovarian and uterine tumors without renal disease, and that albuminuria occasionally occurs early

* Transactions of the Obstetrical Society of London, Vol. viii., p. 323.

in pregnancy, but yields to treatment, while the pregnancy goes on to its natural term.* Dr Barnes conceives the renal affection to result from the kidneys being overpowered in their effort to eliminate the excrementitious matters forming in the system. He thus regards the process as essentially corresponding to what occurs in scarlet fever. Dr Braxton Hicks† inclines to think that the renal affections and the convulsions, which so often co-exist, may both be caused by some deleterious ingredient circulating in the blood. It certainly seems reasonable to suppose, that among the peculiar tissue changes which go on during pregnancy, morbid products may in certain circumstances be evolved which are capable of leading to serious irritations of excretory and other structures.

* Dr Barnes, Transactions of Obstetrical Society of London, 1867, p. 336. † Op. cit

CHAPTER VII.

THE INFLAMMATORY FORM.

Treatment.

THE great objects of medicine being to alleviate suffering, and to obviate the tendency to death, it is clear that, in the treatment of this disease in its acute and early stages, our aim must be to get rid of the dropsy, and to avoid or cure uræmia. In the later stages, again, along with or sometimes without these indications, we have to endeavour to improve the condition of the blood.

Indications of the former class are best fulfilled by increasing the flow of urine; and the best agents for this purpose are diuretics, and dry or wet cupping over the kidneys.

Digitalis is entitled to the first place among the diuretics. It is very efficient, and appears never to irritate the kidneys. It is best given in the form of infusion, in doses of from one to four drachms, repeated several times during the day, according to circumstances. It may also be given as tincture, in doses of from ten to forty minims, or as pill, alone or in combination with squill, or, as some authorities recommend, with squill and a small dose of blue pill. During its administration we must, of course, be watchful of the state of the circulation. This remedy may also be administered

through the skin, cloths steeped in the infusion of the Pharmacopœia, or in a stronger solution, being applied over the abdomen.

Acid Tartrate of Potass is often very beneficial, and may be given with the digitalis. It should be administered in doses of from ten to forty grains several times a day, either as a confection with treacle, or simply suspended in water or butter-milk.

Acetate of Potass, administered in doses of forty to sixty grains, three or four times a day, is a most efficient diuretic, and may be advantageously combined with one or more of the other remedies.

Nitrate of Potass, in doses of twenty to thirty grains frequently repeated, is in general use as a diuretic; and, although in my opinion it is not equal in activity to the other salts above-mentioned, may sometimes be given with advantage.

Oil of Juniper, especially administered by inhalation, is sometimes exceedingly useful, and as it is a remedy which many patients like, it may be given two or three times a day in addition to other medicines. Sir J. Y. Simpson suggested this mode of administration several years ago; and for some years past I have used it, often with great success. It may be administered by dropping some of the oil upon a sponge which has been previously wet with hot water, the vapour passing off carries with it the volatile oil, which may thus be conveniently inhaled, or any of the numerous inhalers now in use may be employed. *Juniper* may also be given in the form of spirit, in doses of from fifteen minims to half a drachm. As this drug appears sometimes to irritate the kidney, its

action must be carefully watched. Gin is a popular and agreeable form for administering juniper.

Spirit of Nitrous Ether is also useful in some cases, given in mixtures in combination with other remedies, in doses of from twenty minims to a drachm.

Infusion of Scoparium is an excellent vehicle for the administration of more potent diuretics, and appears in many cases to aid their action.

Tincture of Cantharides is a good stimulant diuretic, and may be recommended in the later stages of the disease.

Water is not the least important of the diuretics, and the patient should be encouraged to take as much as he conveniently can. Its action has long been recognised, but the profession is indebted to Dr Dickinson, of St George's Hospital, London, for some interesting observations as to its value.*

It is well to administer several diuretics together, as a combination often produces the effect when individual remedies fail.

Dry Cupping over the loins is often followed by marked improvement, especially in the first stage of the disease, when the organs are much congested. It may be repeated several times in a day, and on successive days, on each occasion with renewed benefit.

Wet Cupping, to the extent of from one to ten ounces, according to age and other circumstances, may be employed in urgent cases in which dry cupping fails to give relief. I have seen it, not only relieve the kidneys, but rouse a patient from uræmic coma, and rescue from imminent death.

* Edinburgh Medical Journal, 1864.

Venesection has often been employed with benefit in acute and severe cases occurring in robust individuals, especially when uræmic symptoms prevailed. The best results have often been observed in uræmia occurring in the puerperal states. It may even be repeated with advantage. In most instances, however, local depletion by cupping or by leeches proves sufficient.

Hot fomentations and poulticing over the kidneys are often very useful, particularly in children. They appear to relieve the congestion of the organs.

Cathartics.—Some authors regard these as the most valuable remedies, and certainly their employment is sometimes followed by marked improvement. But several disadvantages attend their use—they interfere with nutrition, their action is exhausting, and they cannot, for any considerable period, be employed to relieve the kidneys of their work. Their great use is, in fact, in meeting temporary emergencies. If the onset of the inflammation be so severe as completely to check the secretion of urine, copious purgation may remove water from the system, and relieve the congested kidney. It is in such circumstances only that I would rely upon them, but in such I have seen them markedly beneficial.

Compound Jalap Powder is the best form of cathartic, and may be given in doses of from half a drachm to a drachm repeatedly. *Elaterium* is praised by some, but appears irritating to the bowels and depressing to the system. It may be given in pill, in doses of from 1-12th to 1-6th of a grain. Perhaps there is more virtue in a good dose of *Castor*

Oil, or of *Sulphate of Magnesia*, than some authors suppose. It must be borne in mind, that while the advantages which some ascribe to cathartics may be problematical, it is important always to keep the bowels free, and tending rather to looseness than to constipation.

Diaphoretics.—With some authors of deserved repute, this appears to be the favourite plan of treatment. *The Vapour or Hot-Air Bath* is often followed by excellent results. But so far as I have seen, while it may be useful in combination with diuretics, it is not itself sufficient to effect a cure in severe cases. The best mode of employing it is the following: The patient being well tucked in, a lamp is introduced under the bed-clothes, and in many cases copious perspiration is speedily induced; or, the patient may be seated in a chair, well covered with blankets. I have seen the perspiration followed by marked increase of discomfort. Much care is needed in order to avoid exposure to cold, as bad results sometimes follow from negligence in this respect. *The Warm Bath* (100° to 102° Fahr.) is in many cases a more convenient method of acting on the skin, and is specially useful in the scarlatinal dropsy of children. The diaphoretic action of baths is greatly favoured by the administration of such fluids as warm tea or gruel.

Of medicinal diaphoretics, the *Solution of Acetate of Ammonia* has appeared to me the most useful, and it may be advantageously combined with the diuretics. Dr Christison recommends Dover's powder in the dose of from five to eight grains three times a day. He says

that, in addition to its diaphoretic action, it is useful as an anodyne and calmative for removing pain, and allaying irritability and restlessness. James' powder has also proved, in his experience, a good diaphoretic.

As to the rationale of these different plans of treatment, the leading idea of those who exclusively employ cathartics and diaphoretics is, that they relieve the system of water and urea, while the kidneys are allowed to rest; and this is deemed by them of great importance, inasmuch as it is assumed that rest is an essential point in the treatment of inflammations, and stimulating inflamed kidneys to increased action is in direct opposition to this general principle. But experience has abundantly proved these views erroneous, and has fully established the value of the treatment which the pathology of the disease suggests. The danger to life results from the occlusion of the uriniferous tubules, obviously then, the clearing out of these tubules must be desirable. If they be well cleared out, the kidney has a much better chance of being restored to a healthy state. I have satisfied myself that, when the old disorganised epithelium of the tubules has been removed a new epithelium is formed; but such a formation cannot take place unless the old be carried away. It thus appears that, even if it were possible to eliminate by the bowels or skin the amount of water, urea, and other urinary ingredients which should naturally be passed off by the kidneys, it would afford no ultimate gain to employ this method, for, the renal tubules remaining obstructed no new epithelium could be produced, the old structures, which were not washed away by streams of urine, would be

slowly absorbed into the system, and tubule by tubule the organ would atrophy. On the other hand, if, by the action of digitalis, or oil of juniper, or cream of tartar, we can lead to a copious transudation through the vascular tufts of the malpighian bodies, we wash away the morbid material which obstructs the tubules, we give the kidney the opportunity of forming new epithelium, and of returning to a normal condition.

In the later stages of the disease, the plan of treatment suitable for the first stage may be indicated, but very constantly hæmatic remedies are required. Iron in all its forms is useful, but especially the tincture of the perchloride, which appears also to possess diuretic properties.

In many cases dropsy may be got rid of by mechanical measures; for example, by pricking the dropsical parts with a needle. The older plan of scarifying is very apt to lead to erysipelatous inflammation, but I have rarely seen any considerable irritation follow the pricking. Water often drains away from such openings in large quantities, and not unfrequently this seems to relieve the kidneys, diuresis becoming much more marked after its establishment. Paracentesis abdominis may also sometimes be required, and followed by much benefit.

On the appearance of uræmic symptoms there is need of prompt action. In my experience, cupping over the kidneys and active purgation have appeared most useful in the cases tending to coma. In cases of a more purely convulsive type the administration of chloroform has proved more beneficial. Bleeding

must of course, as a rule, be avoided in advanced stages of the disease in which the constant drain of albumen has induced anæmia.

In the nausea and vomiting which often accompany the disease, no remedy seems to equal ice, although relief may be occasionally obtained by the use of hydrocyanic acid and other gastric sedatives.

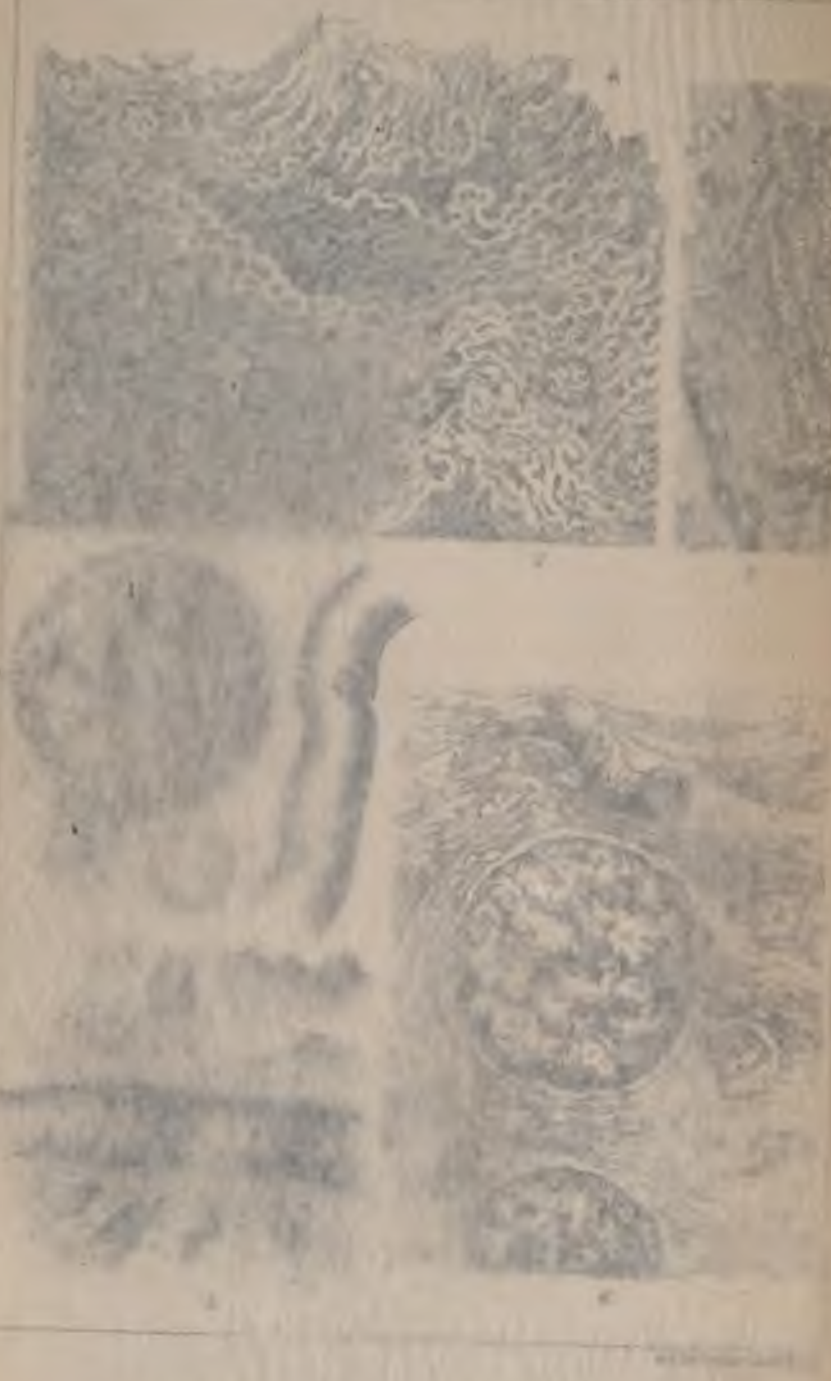
In the catarrhal and dropsical affections of the respiratory passages, counter-irritation and dry cupping have appeared to me useful. The inflammatory complications must be treated with a due regard to the avoidance of lowering remedies. Mercurials, in particular, as tending to impoverish the blood, are distinctly contra-indicated.

The *diet* during the early stages should be simple, consisting of farinaceous substances with milk, chicken soup, beef-tea, and such like, but butcher's meat should be avoided, or given with caution. In the more advanced stages, the diet should be as nutritious as possible, and then, certainly, flesh meat is by no means injurious. A moderate allowance of stimulants is useful in the chronic stages, and perhaps the best form in which they can be given is gin toddy.

Special care should be taken to guard the patients from cold: a flannel night-dress should be worn by those who are confined to bed; and in the more chronic conditions, when patients are able to go out, flannels should be worn next the skin; neglect of such precautions sometimes leads to the most serious results. In the case of patients who can afford to go abroad, a residence in the south of Europe, or even in tropical regions, may be recommended, for it is certain that

the disease is less common there than in this country. It appears, indeed, that the results of such change of climate might, in this disease, be much more satisfactory than they are in cases of phthisis pulmonalis.

PLATE IV.



CHAPTER VIII.

THE WAXY OR AMYLOID FORM.

Morbid Anatomy.

We now proceed to consider the form of Bright's disease which originates in the vessels,—the waxy or amyloid degeneration. For convenience of description, we may divide this affection also into three stages, viz. :—

1*st*, That of simple degeneration of the vessels.

2*d*, That in which a secondary alteration of the tubules is superadded, and

3*rd*, That of atrophy.

As we have seen that in the first form of Bright's disease the inflammation was the primary affection, and that the enlargement, the fatty degeneration, and the atrophy resulted from it, so here we shall find that the peculiar degeneration of the vessels is the primary change, and that the enlargement and ultimate atrophy are but consequences of it.

Before describing the different stages, it may be well to indicate the characters of the changes in the vessels which exist in all the stages. As seen by the naked eye, the affected tissues present the appearance, more or less distinctly, of boiled starch or sago, the appearance which we see most characteristically, because in largest mass in certain forms of the waxy spleen. On microscopic examination, either with low

or high powers, the degenerated parts present a peculiar dim translucency, and are generally more voluminous than natural, while their outlines are somewhat indistinct.—(*Plate* iv.) The iodine test is, however, in my opinion, the most reliable characteristic. When poured over a surface, it everywhere produces a yellowish colour, but the degenerated parts assume a reddish brown, mahogany red, or orange red hue, and stand out very conspicuously.—(*Plate* v.) It often happens that points of degeneration are by this means detected, which could not be made out by mere microscopic examination, however careful. Now and then the further addition of sulphuric acid produces a beautiful blue colour in the degenerated parts, but much more commonly only a purplish hue. As the iodine test is in itself perfectly sufficient, I now rarely add the sulphuric acid. Iodine is not the only colouring matter which is specially absorbed by the waxy material; carmine, indigo, and magenta have all been found to exhibit this property, but none of them is preferable to the test first introduced by Virchow. I do not enter here into the question of the nature of the degeneration, but consider it specially in a supplementary chapter at the end of the book.

1*st. The stage of simple degeneration of the vessels.*—The vessels alone are affected. To the naked eye the organs appear natural, their capsule is easily stripped off. The surface is smooth. In size and weight they are natural. The relative bulk of their cortical and conical parts is normal. The colour, both on the surface and throughout the organ, is natural,

or perhaps a little paler than usual. Only the experienced eye may observe that the malpighian bodies are somewhat too distinct, and present something of the sago-like translucency, but even the most experienced may err or be in doubt until the iodine test or microscopic examination, or both, afford their more positive evidence. The degeneration commences in the cappillary tufts of the malpighian bodies, and in the transverse fibres of the middle coat of the small arteries: most commonly it originates in the cortical substance, sometimes, however, it is more abundant in the straight vessels of the cones, and by no means unfrequently it affects both sets of vessels equally and simultaneously. Wherever it may have begun, it seems to be in general equally diffused throughout the corresponding parts of the organ, and sooner or later it affects most of the small arteries and malpighian tufts. All this may co-exist with little or no abnormality in the tubes. The malpighian tufts appear swollen, and the individual loops more translucent than natural. On the small arteries, there are thickening here and there, the thickened parts presenting the same translucency as is seen in the tufts, and not unfrequently a series of such nodular thickenings occurring along the course of an artery gives it, especially when iodine is applied, an appearance similar to that of Ipecacuan root.—(*Plates* iv. *and* v.)

2*d*. *The stage of transudation into the tubules.*—In this stage the organ is obviously abnormal. It is increased in bulk and weight; its capsule is easily stripped off, and the surface is smooth and pale. A

few stellate vessels ramify on it, and it presents little or none of the mottled appearance seen in the second stage of the inflammatory form. On section the cortical substance is found relatively increased, pale and dense, though not fibrous, and presents much the appearance of white bees-wax. The cones are pink, and of natural size, but the whole cortical substance has the peculiar waxy appearance, and scattered over it are numerous minute semi-translucent points, best seen when the light falls on them obliquely—the malpighian bodies. On examining a section under a low power (50 diameters) we see the malpighian bodies and the arteries degenerated as we have already described, but, in addition, we find many of the tubules full of matter, not dense and opaque as in the inflammatory form, but tolerably transparent. It must, however, be noticed that it neither presents the peculiar translucency nor the coloration with iodine which are characteristic of the waxy degeneration, but exactly resembles the material of hyaline tube-casts.—(*Plates* iv. *and* v., *fig.* 4.) The basement membrane of the tubes, and even the epithelium, are, it is said, in some cases, so changed as to assume with iodine the characteristic waxy coloration. I have frequently looked for this, and have seen the cells present the swollen, dimly translucent appearance, but never the peculiar coloration. The basement membrane I have found thickened and waxy-looking, without any coloration taking place on the application of iodine, and on a few occasions I have seen that coloration. The cells frequently contain numerous minute fatty granules.

What is the cause of the increased bulk of the

organ? To a slight extent the enlargement of the truly degenerated parts must account for it, but it is mainly due to the distension of the tubules in the manner we have described; and this distension of the tubules most probably arises from the transudation of the fluid parts of the blood through the degenerated walls of the vessels. A slow transudation of fibrine most probably takes place, this coagulates in the tubules, and so we have a condition closely resembling that which occurs in the inflammatory form. This explains, too, the fatty degeneration of the cells. It is to this stage that the term "waxy kidney" is most applicable.

3*d*. *The stage of Atrophy.*—In this stage the organ is reduced in bulk and weight. The capsule may be torn off without much difficulty. The surface is uneven, rough and granular, of a pale waxy colour, but also occasionally mottled here and there with sebaceous-looking material. On section, the cortical substance is found much diminished, while the cones are nearly natural. The malpighian bodies are large, prominent, closely grouped together; the tubular structures are wasted; the smaller arteries are dilated, and their walls thickened. On examining a thin slice under a low power, we find the relative increase of the vascular elements very remarkable.—(*Plates* iv. *and* v., *fig.* 1B.) In some parts, and in extreme cases, I have seen the malpighian bodies so closely grouped together as to remind one of a bunch of grapes, the degenerated artery representing the stem. Tubules here and there continue distended, but most are atrophied,

their walls collapsed, and represented only by fibrous tissue.—(*Plates* iv. *and* v., *fig.* 6.) The degree of atrophy varies in different instances from about the natural size of the organ to a fourth, or even less.

As to the time required for these changes it is difficult to speak, for I have not yet traced a case from its commencement to its fatal termination, and I am not aware that any case has been so observed. The early symptoms are often so little attended to that it is difficult to make out the date of origin of the disease in cases which come under observation. I know of one case which has been going on for more than eight years, and does not appear to be approaching a fatal termination. I examined the kidneys in another case, which had been under my observation for three years, and they were not much smaller than those of a healthy individual. And in another fatal case, I found that distinct symptoms had existed for about six years, and yet the organs were less atrophied than I have frequently seen them.

CHAPTER IX.

THE WAXY OR AMYLOID FORM.

Clinical History.

An individual who has long suffered from wasting disease, such as scrofula, caries, necrosis, or syphilis, or who, though without palpable disease, is of feeble constitution, feels an increasing weakness, and begins to pass large quantities of urine, and to drink largely. He is, contrary to his usual custom, obliged to rise repeatedly during the night to make water, and on each occasion passes a considerable quantity. The amount of urine varies from 50 to upwards of 200 oz. daily, always bearing a relation to the amount of fluid drank, generally nearly equalling it in amount, sometimes even exceeding it. The feet and ankles may become œdematous after a hard day's work, but return to their natural condition during the night's repose. In many cases there is observed a hardness and swelling in the hepatic and splenic regions, dependent on an increase of bulk of the liver and spleen. The patient feels a constant lassitude and unfitness for exertion. His urine gradually becomes albuminous, and a few hyaline tube-casts are to be found in the very scanty sediment which it throws down. It is of low specific gravity, 1005 to 1015. The blood in many cases presents some peculiarities when examined

microscopically, the white corpuscles being somewhat increased in number, and the red presenting a flabby appearance, with a marked tendency to tail—that is to say, instead of forming into rouleaux, like healthy corpuscles, become stretched out into a series of long oval bodies. These changes I have observed only when the degeneration affected the lymphatic or blood glands. The patient may continue in this state for months, or even years,—may undergo a temporary or perhaps even a permanent improvement—the liver and spleen becoming diminished in bulk, and the blood resuming a more healthy character. But, in a great majority of cases, no such favourable result occurs, the patient, sooner or later, becomes steadily worse, and sinks, either from the renal disease, or from one of the many maladies which accompany it.

If from the renal disease ascites or general dropsy gradually supervenes, the urine may diminish in quantity, so as at times to be almost or altogether suppressed. It is very albuminous, but not of high specific gravity, and contains fatty and hyaline casts. Occasionally drowsiness comes on, and the disease terminates amid coma and convulsions. In such cases it is, however, as a rule, found that an inflammatory affection of the tubules has become superadded to the original degeneration of the vessels, and so the death cannot properly be referred to the waxy degeneration. But, in other instances, a fatal termination results from the accompanying maladies, perhaps most frequently from diarrhœa, a result of waxy degeneration of the intestine, from phthisis, one of the most common causes of the lesion, from ex-

hausting discharges of chronic abscesses, or of caries, or necrosis, or from constitutional syphilis.

The renal disease is thus met with in the post-mortem theatre in many different stages; for though itself by no means ranking high as a fatal malady, those with which it is associated are so eminently fatal as to give the pathologist only too many opportunities of studying the disease in all its phases.

As examples of this form of the disease, we may take the following cases:—

Cases illustrative of the First Stage.

Case XV.—*Waxy degeneration of kidneys, spleen, intestines, and liver; tubercle of lungs, lumbar abscess, &c.*—J. C—, æt. 14, a deformed boy, was admitted under my care to the Royal Infirmary in January 1866, on account of lumbar and psoas abscess, with tubercle of the lungs. He passed from seventy to one hundred or more ounces of urine daily. At first it contained no albumen; afterwards slight traces were found, although by no means constantly. In April he became affected with exhausting diarrhœa, at times dysenteric in its character; and in consequence of this, and the wasting discharge from the abscess, he died exhausted on May 7th. He never had a trace of dropsy.

Autopsy.—The body was much emaciated and deformed. There was an opening in the abdominal parietes close to Poupart's ligament, and another in the lumbar region on the right side. The abscess was connected with caries of the bodies of several of the dorsal vertebræ. The lungs contained some obsolete tubercles. On the mitral valve there were some vegetations. The liver was large, fatty and waxy. The spleen was bound down by numerous adhesions; its malpighian bodies and small arteries were waxy. In the former there were numerous extravasations of

blood. The intestines were waxy. The kidneys were small—in some parts atrophied. In some of the tubules there was slight evidence of inflammation; in many there was a finely fatty condition of the cells, but there was no free exudation into the tubules, many of which were normal. The malpighian bodies and the arteries of the cortical substance, as well as the arteries of the cones, were waxy.

Commentary.—In this case we had the waxy degeneration originating in one of its most common causes—a wasting discharge from a carious bone, and manifesting itself (so far as the kidneys were concerned) by only one symptom, viz., the increased flow of urine; but so reliable do I consider the symptom when associated with any of the recognised causes of the waxy degeneration, that I ventured to anticipate, on his first admission to the hospital, the appearance of albuminuria, an anticipation which was not disappointed. The puckerings on the surface of the organs were, I think, results of slight local inflammatory action, too local to induce general symptoms.

CASE XVI.—*Waxy degeneration of kidneys, tubercle of lungs and intestine, constitutional syphilis.*—J. M—, æt. 24, a man of syphilitic constitution, was admitted to the Royal Infirmary, under the care of Dr Scoresby Jackson, with symptoms of incipient phthisis, on March 8, 1865. His tubercular disease gradually became worse, and became associated with colliquative diarrhœa. He died exhausted July 7.

Autopsy.—The body was emaciated; the heart thin and feeble, the valves natural. Both lungs contained tubercular deposit, and were riddled with cavities. The liver weighed 4 lb. 6 oz., was waxy and fatty. On its surface there were a number of small cicatrices and adhesions to neighbouring parts. The spleen was waxy. In the intestines, particularly in the sigmoid flexure of the colon, there were numerous tubercular ulcers. The kidneys

were somewhat pale; they weighed together 10½ oz. The vessels of the cones and some of those of the cortical substance were slightly waxy. The tubules were natural.

Commentary.—This case, occurring in a syphilitic individual affected with pulmonary and intestinal tubercle, proved fatal from those affections before the waxy disease of the kidneys was far advanced. I was unable to obtain any information as to the amount and quality of the urine, but it is probable that, from the severe diarrhœa, the quantity could not have been much increased.

CASE XVII.—*Waxy degeneration of kidneys, tubercle of lungs, &c.*—A. M— was under treatment in the Royal Infirmary, under the care of Prof. Laycock, for tubercle of the lungs. She died exhausted. On post-mortem examination the body generally was emaciated, and there was slight œdema of the labia. The heart was slightly dilated. The pleural surfaces were adherent, and the lungs contained much tubercle. The liver was enlarged and waxy; weighed 5 lb. 7 oz. The spleen was waxy. The kidneys were of natural size; the tubules were natural, the vessels in a state of waxy degeneration. The villi and vessels of the small intestine were waxy.

Commentary.—In this case also the renal symptoms were not recognised during life, attention having been mainly directed to the pulmonary affection.

Cases illustrative of the Second Stage.

CASE XVIII.—*Waxy degeneration of kidneys, syphilis, diarrhœa, &c.*—A. C—, æt. 30, was admitted to the Royal Infirmary, under the care of Dr Sanders, May 30, 1864. She stated that she had enjoyed good health until within four weeks of her admission, but

for some months before she had observed that she passed a larger quantity of urine than natural. She was obliged to get up several times during the night to micturate. She had a little dropsy, but it disappeared on the occurrence of diarrhœa, a few weeks before admission. Her urine was pale, of sp. gr. 1010, contained much albumen, always exceeded 60 oz. daily, although she was affected at the same time with severe diarrhœa. She had frequent vomiting, and gradually became exhausted, and died June 28. Her family was strumous. There was no positive evidence of syphilis, but she had a cachectic appearance, and complained much of pain in her bones.

Autopsy.—The body was somewhat emaciated; the heart and lungs were natural. The bronchi contained much muco-purulent fluid. The liver was large, weighed 4 lb. 6 oz., was bound to the diaphragm by numerous old adhesions; it was fatty and waxy thoughout; both the cells and vessels were waxy. The spleen weighed 1 lb. 1 oz.; was extremely waxy. Both kidneys were enlarged, the left weighed 9½ oz., the right 7½ oz. The vessels were extremely waxy, both in the cortical substance and in the cones. The tubules were in many parts distended with a clear hyaline material, and the epithelium was in many parts finely granular. The basement membrane of the tubes also appeared in some parts waxy. The intestines were waxy.

Commentary.—This case proved fatal during the second stage of the disease, not from affection of the kidneys, but from the severe diarrhœa, which resulted from the disease of the intestines. The disease which, as we have seen, is insidious in its progress, had come on at least six months before her death, but it is very probable that it had existed even before that time. Notwithstanding the severe diarrhœa, she passed an excessive quantity of urine during the whole time that she was under observation.

CASE XIX.—*Waxy degeneration of kidneys fatal in second stage; struma.*—M. M—, æt. 17, was admitted to the Royal Infirmary,

March 3rd, 1860, and was under the care of Drs Bennett and Laycock. She had had carious disease of the vertebræ. During February she had noticed that she was making more water than usual. Her urine was always albuminous, ranged from 50 to 120 oz. daily, and deposited hyaline casts, which contained here and there a fatty cell. In May she had diarrhœa, and at the same time dropsy; her urine diminished to 30 or 40 oz. daily. It contained more tube-casts, and in the casts there were more numerous cells than formerly. She died in the beginning of June.

Autopsy.—The body was somewhat dropsical. The liver and spleen were waxy. The kidneys were large and pale; exhibited the characters of the second stage of the waxy with some degree of the inflammatory affection of the tubules. There was no ulceration of the intestines.

Commentary.—This was a very typical case of the waxy disease, both in respect of previous history and of symptoms. The patient died four months after she had noticed the increased flow of urine, but it may have existed much longer. She did not come under observation until the albumen had appeared. Some degree of inflammation was superadded to the original malady, and induced the dropsy and diminution of the urine.

Case XX.—Edward Burns, a labourer, æt. 30, married, resident in Edinburgh, was admitted to Paton's Ward, January 12, 1860. Patient states that he has had very little sickness, and, in particular, never had syphilis; but he confesses to having suffered from bubos, resulting from a strain; and his prepuce is remarkably contracted, and his throat presents most syphilitic-looking ulcerations.

On admission, his throat was ulcerated, his voice was husky, and he had a harsh cough, with occasional muco-purulent expectoration. At the apex of the right lung there was harshness of respiration, but no increase of vocal resonance; cardiac sounds normal; pulse 80, small and feeble. Blood poor in corpuscles;

the white relatively more numerous; the red pale and flabby, with a tendency to tail, and form into rows like a string of beads rather than a rouleau of coins. Tongue clean; appetite pretty good; bowels open. Hepatic dulness extends from the sixth rib to the umbilicus. The spleen is also considerably enlarged. The urine is highly albuminous, of low specific gravity, and contains a few hyaline tube-casts. Patient stated that he never observed anything particular about his urine; but on its being measured, it was found to amount to upwards of 100 oz. daily. It was always of low sp. gr., and never contained a trace of sugar. There was no œdema of the legs, unless occasionally, when he had been working hard, and then his ankles got swollen at night. He continued under treatment for about four months, during which his general health improved, and his liver diminished slightly in size. The amount of urine became somewhat lower, and his blood presented a more healthy appearance. He was dismissed, at his own request, on April 30th; and thereafter was able to work at his business for some time.

7th April 1861.—His general appearance is better than it was last year. He states that he is quite well, but that the daily amount of urine has not diminished. It is highly albuminous, of low specific gravity, and contains casts. His tongue is clean; his appetite good; his bowels are moved twice a-day. The liver is much enlarged, measures eight inches vertically in the right mammillary line, and extends considerably across the epigastrium to the left side. The spleen is also enlarged. The blood contains an excess of white corpuscles. Expiration is harsh and prolonged at the apices of both lungs. The heart-sounds are altered in tone, but not of a blowing character.

13th August 1862.—The patient again presented himself, and the following notes were taken:—He is more emaciated. States that from increasing debility he has been unable to work for a month past. He still makes large quantities of urine, which is albuminous, but not so intensely as before. It deposits a sediment containing hyaline tube-casts, with oil-granules here and there arranged in groups, as if resulting from disintegration of cells. There has been no dropsy of late. The liver, though still enlarged, is decidedly diminished since last report. He complains much of his breathing.

22*d September* 1863.—He complains much of difficulty of breathing, and of cough and headache when he attempts to stoop; he has also dropsy; and from all these symptoms feels himself unable to follow his usual work. The amount of urine is still large. He is obliged to rise three or four times every night in order to micturate. The urine is albuminous, and contains casts. He entered the Infirmary, and under the care of Dr Sanders improved so as to be able to go out, and for a time pursue his usual avocations; but in November he again presented himself complaining of a further aggravation of his symptoms. He died exhausted soon after readmission, in November 1863.

Autopsy.—The body was somewhat emaciated. The heart was enlarged; its left side was much hypertrophied. The aortic valves were competent; but at the base of one of the segments there was a calcareous mass. The aorta was very atheromatous. The lungs were very œdematous; the bronchi were congested and full of mucus. The liver was about the natural size. On its surface were a number of nodules and cicatrices. At the bottom of some of the cicatrices nodules of a pale colour were visible. On section numerous nodules were found scattered throughout the organ; they were pale, dense, and had an appearance exactly resembling bees-wax; their structure was much denser than that of the surrounding tissue. In some nodules there were streaks of fibrous tissue throughout the substance and round the margin, and the greater the proportion of that tissue the deeper were the cicatrices. In the nodules elevated above the surface there were no such streaks, or very few. In those situated at the bottom of deep cicatrices, the fibrous element was abundant, or even in excess of the glandular. On applying iodine to these masses, the whole of the waxy-looking material assumed the brownish red colour characteristic of the amyloid degeneration, but the fibrous streaks simply assumed a yellow tinge. Microscopically, the masses were found to present exactly the characters of ordinary amyloid hepatic cells. They were composed entirely of these cells, enlarged, transparent, and finely granular. In some parts the cellular elements were broken down, and a finely granular material, containing some oil globules, was present. The fibrous tissue in the masses presented the ordinary characters of connective tissue; and where it was most abundant the cells were most atrophied.

Throughout the rest of the organ the cells were little affected with the waxy degeneration, but some of the small vessels showed it distinctly. The fibrous bands were seen passing into the tissues round the cicatrices and nodules. The capsule of Glisson was thickened in some parts, and on applying the iodine externally to the cicatrices no reaction was observed. The spleen contained one cicatrized mass, which presented no reaction with iodine. The kidneys were somewhat contracted in the cortical substance, and presented a very well-marked instance of the amyloid degeneration of the vessels and malpighian bodies. Some of the tubules contained hyaline material, which did not become coloured with iodine. There was some degree of amyloid degeneration of the villi of the small intestine; the bowels were otherwise natural. The prepuce presented traces of the old syphilis, and it had been previously ascertained that there were numerous syphilitic ulcerations in the throat.

Commentary.—This case was evidently of considerable standing. Notwithstanding his denial of it, I was satisfied from the first that he had been the subject of syphilitic disease, and in the course of his illness he acknowledged that it was so. The case affords an excellent example of the latency of the symptoms in many of these cases of waxy degeneration. The condition of the urine was eminently characteristic of the disease; and but for the indications it afforded, it would have been impossible for us to have diagnosed the existence of the affection. There was no characteristic appearance of the countenance, no diarrhœa, no certain history of old-standing disease by which we could have been led to the opinion. It proved fatal after it had lasted upwards of three years, and the kidneys had begun to atrophy.

Cases illustrative of the Third Stage.

CASE XXI.—*Waxy degeneration of kidneys fatal in third stage, &c.*—J. P—, æt. 50, a quarryman, resident in Edinburgh, was admitted under my care May 12, 1865. He denied having had syphilis, and stated that he had been in general healthy. For some time before his admission he noticed that his feet became swollen at night, that he had frequently to get out of bed in order to make water, and that he had great thirst.

On admission.—His legs were œdematous. His complexion was good ; his cardiac sounds were natural, his arteries atheromatous. There was a slight arcus senilis. The red corpulses of his blood were flabby. He had some bronchitis. His appetite was impaired. His bowels acted naturally, his liver and spleen were of normal size. The urine averaged about 120 oz., was pale, of sp. gr. 1008, distinctly albuminous. No casts were found. Under treatment he improved, and was dismissed June 5th.

On the 12th of that month he returned, feeling decidedly worse. He then had some muscular twitchings, without loss of consciousness. These were followed by a severe convulsion on the 14th, during which he became quite unconscious. In the afternoon he had another severe fit. He gradually became comatose, and died on the 22nd.

Post-mortem examination twenty-three hours after death.—The body was not dropsical nor much emaciated. The heart weighed 1 lb. 3 oz., was dilated and hypertrophied. There was considerable atheroma of the arteries. The lungs were congested, œdematous, and emphysematous ; contained traces of old tuberculous or syphilitic deposit. The liver weighed 4 lbs. Many of its cells were fatty, and its smaller vessels were waxy. The spleen weighed 6 oz., was not waxy. The kidneys were waxy. They were considerably reduced in size, the capsule stripped off without much difficulty, the surface was rough and granular. The malpighian bodies were closely aggregated, especially close to the surface. The tubules were in that part atrophied, represented merely by fibrous tissue. The vessels and villi of the intestine were waxy, but the cavity contained some hardened faces. In the substance

of the brain, in the white matter of the posterior lobe of the right hemisphere, there was a recent clot of the size of a pea; the substance generally was congested and œdematous.

Commentary.—This case was also of considerable standing. The patient had evidently had syphilis. His case affords another example of the latency of the symptoms in the waxy degeneration. The condition of the urine was eminently characteristic, and but for the indications it afforded, we could not have diagnosed the existence of the affection, there being no characteristic appearance of the countenance, no enlargement of the liver, no diarrhœa, no certain history of old-standing disease by which we could have been led to the opinion. The nervous symptoms which preceded death were probably due to cerebral congestion and apoplexy, rather than to blood poisoning.

CASE XXII.—*Waxy degeneration of kidneys fatal in third stage; syphilis, uræmia.*—J. N—, æt. 37, a miller, was admitted to the Royal Infirmary, under my care, on May 13th, 1865.

Previous history.—Twelve years before admission he had contracted constitutional syphilis, and for eight years had been out of health. He suffered from various constitutional symptoms, and in 1862 he found that he was obliged to rise frequently during the night to make water. The daily amount of urine passed was found at that time to be from 150 to 180 oz.

His symptoms had gradually become worse; he had cough and some difficulty of breathing, and once, in March, 1865, he had a fit, which lasted for three minutes, and during which he was unconscious and moved convulsively.

On admission.—The complexion was generally fair; the cheeks and nose were red from dilatation of small vessels. The tongue was clean, the appetite good. The liver measured 6½ inches vertically in the mammillary line. Heart sounds were natural. Some of the smaller vessels were atheromatous. The white cor-

puscles of the blood were somewhat increased in number, the red were soft and flabby. The nervous system was natural. The amount of urine ranged from 90 to 150 oz. daily; it was pale, of sp. gr. 1010, albuminous, and contained some hyaline and granular casts. By careful measurement it was ascertained that the amount of fluid consumed daily was less than the urine passed.

Early in June he became affected with diarrhœa; he also became peculiarly irritable, had distressing dreams, and fancied he saw black objects, particularly a large black rat, flitting about the ward. He also complained of pain in the left forehead. He gradually became exhausted and more delirious; he had a good deal of twitching of the muscles, but no convulsion, and died June 14th.

Post-mortem examination, twenty-six hour after death.—The body was not dropsical nor emaciated. The pericardium contained a little fluid. The substance of the heart was pale and fatty. The valves were competent, but there was some deposit at the bases of some of the segments. The bronchi were congested, and contained a good deal of mucus. Both lungs contained syphilitic deposits, partially softened. The liver weighed 7 lb. 11 oz. It was connected by adhesions with neighbouring organs. It also contained some syphilitic and waxy masses, which evidently bore a relation to the cicatrices. Many of the small vessels were waxy. Many of the cells were waxy and many fatty. The spleen was much enlarged, waxy, weighed 3 lb. 3 oz. It contained peculiar cicatrices and deposits of altered blood. The suprarenal bodies were waxy. The kidneys were large, their surface granular, the increase of bulk being from syphilitic deposits. The vessels of the cortical and conical parts were extremely waxy. Some of the tubules contained fatty matter. The right testicle contained a syphilitic deposit. The intestine was waxy, and was ulcerated at one or two points. The brain was congested and œdematous, contained no syphilitic deposit, and its vessels were not found to be altered. The cord was in the same condition as the brain.

Commentary.—This was a typical case following upon syphilis. The increased flow of urine had been observed for three years, and all the symptoms of the

disease were well marked. The peculiar variety of uræmia which preceded death was also very interesting.

CASE XXIII.—E. H., a washerwoman, æt. 43.—She had been of intemperate habits, but was not known to have had syphilis; she had long-continued polyuria; her urine was of low specific gravity, very albuminous, and contained hyaline casts. She had also a lesion of the aortic and mitral valves. She was dismissed from the Royal Infirmary in May 1860. In November of that year I found that her symptoms were not materially changed. She continued to make from 180 to 210 oz. of urine daily. It was of low specific gravity; it contained albumen and casts. There was slight œdema; the diarrhœa less intense than formerly; the cardiac symptoms unchanged. Throughout the years 1861-62-63 I saw her frequently in St Cuthbert's Poorhouse, in the Royal Infirmary, in the Dispensary, and elsewhere. Her renal symptoms were little altered. A distinct aneurismal dilatation had gradually developed itself; and a certain amount of œdema of the limbs occasionally appeared. When she was last in the Infirmary I had the opportunity, by the kindness of Dr Laycock, of making the following notes:—

20*th April* 1864.—The skin is pale; conjunctiva clear, slightly œdematous. There is a good deal of congestion over the malar bones. The legs are œdematous; the tongue is clean. She has some difficulty in swallowing, particularly solids. Sickness follows eating, and she occasionally vomits. Vertical hepatic dulness in the right mammillary line measures about 5 inches. The bowels are loose. She complains of pain in the left hypochondrium. She has occasional giddiness, and sleeps badly. Her pupils are equal. There is a double blowing murmur at the apex of the heart, and at the base of the neck there is a very distinct aneurism. The urine is copious, exceeding on an average 100 oz. daily. Its specific gravity is about 1008. It is of an acid reaction, contains much albumen, some phosphates, and epithelial and granular casts. Her dyspnœa, dropsy, and general debility gradually increased, until the 5th of June 1864, when she died.

Autopsy, fifty-eight hours after death.—The body was well nourished. The right pleural cavity contained about half an ounce of clear serum. The left pleura was obliterated by old ad-

hesions. Both lungs were congested and œdematous; in several parts there were small dense nodules, whose nature was not determined. The pericardium contained a little fluid, and some lymph was deposited on both its layers. The heart was enlarged, weighed 8¾ ounces; it was fatty. The margins of the mitral valve were thickened. The aortic valves incompetent. The aorta was dilated, its coats sclerotic and atheromatous, and contained some calcareous plates. There were distinct dilatations in the course of the innominate and subclavian arteries. The liver weighed 3 lbs. 3½ ounces, was soft and fatty, and presented no reaction with iodine. Spleen weighed 3¾ ounces, and was not waxy. The right kidney was small, weighing 4 ounces; the capsule was adherent; the surface granular; the cortical substance was atrophied. The left weighed 6 ounces, was distinctly waxy and fatty, less atrophied; the capsule was also adherent. The malpighian bodies, as well as the arteries of the cortex and of the cones, were in a state of waxy degeneration.

Commentary.—In this case there was no history of syphilis, nor of any other exhausting disease. The symptoms which led me to diagnose waxy disease were the polyuria, the albuminuria, the diarrhœa, and the absence of dropsy. Before death there was considerable dropsy, which depended partly on the cardiac and vascular lesions,—partly on the superadded inflammation of the tubules.

Case of many years standing, still under observation.

Case XXIV.—Archibald March, æt. 29, a shoemaker, married, resident in Edinburgh, was admitted to Paton's Ward, February 15th, 1860.

In April 1859 patient was in the Infirmary on account of enlargement of the liver and spleen, with slight leucocythæmia. He was dismissed considerably relieved; but having felt, of late, great oppression on taking food, with occasional bloody vomitings and

increasing general debility, he was readmitted. States that, some years since, he had syphilis, which was followed by eruptions, nodes, &c., and ultimately by the symptoms of which he now complains.

Symptoms on admission.—His general appearance is cachectic and sallow; his chest covered with brownish patches of pityriasis nigra, which have existed for some years. There is no œdema. Pulse is full, 82 per minute. Cardiac dulness 2½ inches transversely. There is a soft blowing murmur with the first sound, loudest at the base. There is a slight relative increase of the colourless corpuscles of the blood, and the red corpuscles have a tendency to tail. Tongue is moist; appetite not good; thirst great. He vomits occasionally after eating. Bowels constipated. The liver measures 9 inches in a line vertical to the nipple; and there is great tenderness on pressure over the whole area of dulness. The splenic dulness, laterally, is 5½ inches from above downwards. Urine was of a pale amber colour; specific gravity 1009; no albumen. Such was his state on admission. He remained for some time under observation; and on March 3d it was ascertained that his urine amounted to 110 ounces daily, and it continued at a similarly high standard, sometimes falling as low as 90 and rising as high as 130 ounces. Finding the amount of urine so large, and the general symptoms so closely resembling those I have just read, I ventured to anticipate the appearance of albumen in the urine. It was carefully tested day by day, and about the 10th of March a trace of albumen was observable. It steadily increased in amount; and, soon after its appearance, a very few waxy or hyaline casts were to be detected by the microscope. Notwithstanding the increased flow of albumen, the patient, under a tonic treatment, with liberal diet, so far improved as to be able to leave the hospital, to resume work, on March 26th.

December 3.—His complexion sallow and cachectic as before; abdomen free from tenderness; liver measures, in line of right nipple, 7½ inches, and the spleen barely 5 inches at the side. He does not know exactly how much water he makes daily, but thinks it is less than when he was in the Infirmary. It is distinctly albuminous. No dropsy. The glands of the neck on both sides have become enlarged within the last ten days. The blood is in the

same condition as formerly. Thus it is evident that, except in regard to the kidneys, considerable improvement had taken place.

4th February 1861.—The liver and spleen have further diminished in size. His appearance is somewhat less cachectic. For some days he has had a pain in the neighbourhood of the umbilicus, and along the margin of the liver, aggravated on movement or on pressure, and after eating. The stools are of a dark colour, and contain some bright red blood. He has no piles. The amount of urine continues high, about 120 oz. daily. It is albuminous.

8th October 1861.—The patient again presented himself; his cachectic appearance is increased; he complains of a severe pain in the lumbar region, and along the spermatic cords. His renal symptoms continue unchanged, and the liver is still distinctly enlarged.

1st June 1864.—Since the last note he has frequently presented himself at the New Town Dispensary and elsewhere; has repeatedly been an inmate of the Royal Infirmary; has been able occasionally to work at his occupation of shoemaking, and has of late acted pretty constantly as cook to the Mid-Lothian Militia, stationed at Dalkeith. The last note I have taken of his case was on 21st March 1864. He continues to make large quantities of water daily, usually upwards of 120 oz. It is still albuminous, but no tube-casts have been discovered for some time. The hepatic dulness is diminished to about six inches; the organ is still painful on pressure. He has no nausea, and his bowels are regular; but on several occasions lately he has had intense diarrhœa, sometimes bloody, and has vomited blood-coloured matters. His complexion is even darker than before, and his eyelids are more œdematous than I have ever observed them. From some observations made for me by Mr Taylor, it appears that his temperature is somewhat lower than natural.

He has since that time been frequently under my care in the Infirmary. The following reports describe his condition when last seen.

17th March 1868.—Again presented himself, complaining of gastric symptoms, which he ascribes to an afternoon's indulgence in whisky. Complexion much less sallow, lips have more colour, tongue somewhat furred, no tenderness on pressure of abdomen, liver measures 5½ inches, splenic dulness 3 inches, cardiac sounds

natural, bowels natural. Thinks he makes about 100 oz. of urine daily. Sp. gr. 1006. Feebly acid reaction, contains a considerable amount of albumen, about one-fourth. Tube-casts scanty, large and hyaline. Blood presented a slight excess of colourless corpuscles. Patient has been working steadily for five months.

30*th March* 1868.—Feels much better. Quantity of urine as usual, albumen is still considerable, about one-fourth.

Commentary.—I have watched this case carefully for between eight and nine years. I was led to anticipate the appearance of albumen in his urine, by the history of syphilis, the enlargement of the liver and spleen, the altered state of the blood, and the polyuria. During all these years the albuminuria has continued, and the urine has been very copious. There has been no dropsy, but the liver, the spleen, and the blood have distinctly improved. His general health is now much better, and I hope that he may further improve. In the narrative of the case little has been said of treatment. It has consisted mostly of iodide of potassium, iodide of iron, good nourishing diet, and attention to gastric, intestinal, and other complications as they arose.

In many cases a peculiar cachexia exists, the characteristics of which are apparent at all stages of the disease. There is a pale anæmic appearance, with occasionally a little dark pigmentary matter in the skin, particularly about the eyelids, an air of general debility, and a pasty or waxy complexion. This would seem to be most commonly associated with the syphilitic form. In other cases there is a

characteristic appearance of the face, with which I have become familiar, when the surface generally is pale and clear, but a very distinct congestion exists over the cheeks. This is not a congestion like a blush, but is seen by the naked eye to depend upon the distention of small vessels quite above the size of capillaries.

The character, then, upon which we mainly depend for diagnosis are the increased flow of urine, the albuminuria, the absence of dropsy, the previous history, the complications, and the appearance of the patient.

CHAPTER X.

THE WAXY OR AMYLOID FORM.

Nature of the Symptoms.

THE symptoms of the waxy kidney which specially deserve attention are those connected with the urine, the dropsy, and the affections of the nervous system.

I. The *urine* is, as a rule, increased in quantity. It ranges from 60 to upwards of 200 oz. daily.* Of all the cases which I have observed in the Royal Infirmary and elsewhere, in none was this symptom absent excepting when severe diarrhœa, or inflammation, or other disease of the kidney, co-existed with the malady. Along with the polyuria there is increased thirst, but careful measurements in many cases have shown that the fluid passed is equal to or in excess of the total amount consumed.

This symptom appears referable to the degenerated state of the vessels. It may be that the degeneration of the muscular fibres of the small arteries leads to paralysis and dilatation of these vessels, but certainly we are entitled to assume that the degenerated walls, although thickened, permit undue transudation of their contents. In the intestine we find this degeneration

* Rosenstein contradicts my statement that the urine is always above the natural amount in this disease; his mistake arises from his failing to distinguish between simple waxy and the combined waxy and inflammatory diseases. I have been much interested to observe, while these sheets were in the press, that Dr Harris of London had recognised the existence of polyuria in this disease in 1860.

accompanied by a corresponding symptom, diarrhœa, often profuse and watery. To this increased permeability of the vascular walls, then, the symptom may be best referred. It has indeed been suggested that the increased flow may be due to increased blood pressure on the unaffected vessels, a consequence of obstruction of the circulation in those which are degenerated; but this, though it might account for the urine being little diminished, seems incapable of explaining an increase. Besides, polyuria may co-exist with very general degeneration of the vessels.

Is this symptom, polyuria, to be regarded as an unfailing evidence of the waxy degeneration? Certainly not: for apart from diabetes mellitus we observe the same symptom in diabetes insipidus, in many nervous diseases, and sometimes in cases of irritation of the kidney from renal calculus. But in all such instances there is little chance of mistake, seeing that they are unaccompanied by albuminuria. In the contracting form of the disease, however, the two symptoms occasionally co-exist, although, as we shall see, not very often.

Is it always present in the waxy degeneration? I have never found it absent, except in cases accompanied by severe diarrhœa, or by inflammation of the tubules of the kidneys, or by a peculiar syphilitic deposit in the stroma of the organ.

In *colour* the urine is generally pale, from the large amount of water it contains. Its specific gravity is, for the same reason, low, from 1005 to 1015.

With regard to the natural solid constituents, we are indebted to Rosenstein and Dickinson for a num-

ber of careful analyses. They found that throughout the greater part of the duration of the disease the urea falls little, if at all, below the natural amount. But it is obvious that in advanced stages, when many of the uriniferous tubules are occluded or atrophied, the quantity must be somewhat diminished. The amount of uric acid varies in different instances. The phosphoric acid is diminished, Dr Dickinson says, more constantly in this than in either of the other forms. Sulphuric acid, chlorine, and alkaline and earthy salts are also diminished.

Albumen is the most important abnormal constituent. The cause of its occurrence here is the increased permeability of the walls of the vessels. At first the degenerated structures permit merely of an increased transudation of water, but as the disease advances albumen also escapes, at first in very small quantities, afterwards in greater amount, sometimes when it has been solidified, equalling in bulk one-fourth, or even one-half, the urine. Blood sometimes also escapes from rupture of the capillaries, but this is comparatively rare.

Tube-casts are, as a rule, not very numerous in this disease, but may generally be discovered by careful examination. They are for the most part hyaline and finely granular; sometimes they contain fatty cells. I have never seen the casts present the peculiar reaction with iodine, although I have occasionally seen appearances somewhat like it. The formation of the casts depends upon transudation of fibrine through the degenerated walls of the vessels, and its coagulation within the tubules.

II. *Dropsy* does not, as a rule, occur in this disease in its early stages, and often it does not appear even when the malady is far advanced. In a series of fifty cases which I examined post-mortem, it was present only in three, that is in 6 per cent; while in a series of cases of combined waxy and inflammatory it occurred in 47 per cent. But in a certain proportion of cases it does appear, and then, if not dependent upon complications, it may be referred to the anæmic condition which the disease, if long continued, induces.

III. *Nervous symptoms* are not so frequent in this as in the other forms of renal disease. They occur only in the advanced stages, and in the cases in which inflammatory disease of the tubules has been superadded.

The character of these symptoms is also at times peculiar, as was seen in the case of J. N., p. 83. The theories of uræmia having been sufficiently explained in a previous chapter need not be considered here.

CHAPTER XI.

THE WAXY OR AMYLOID FORM.

Complications and Causes.

In considering such a disease as the waxy degeneration of the kidneys, it is convenient to group together the causes and the complications, as, from the chronic character of the disease and its causes, they are apt to co-exist.

We shall take up—1st, The Causal; 2d, The Concomitant; and 3d, The Consequent Complications.

A. Causal Complications.

1*st, Tuberculosis of Lungs and other organs.*—Tubercle of the lungs existed in about one-half of all the cases of waxy kidney which I have examined in the Infirmary, tubercle of the intestine in about 18 per cent. In a very considerable proportion of these cases it appeared that tuberculosis was a cause of the renal disease. When we analyse the relationship more closely, we find that tubercle of the lungs was specially associated with the earlier stages, for of those fatal in the first stage it was present in 66 per cent, of those in the second in 60 per cent, and of those in the third in 35 per cent. When we compare this with what we find in other forms of renal disease, the relationship between tubercle and the waxy degeneration becomes manifest, for with

the inflammatory it was present only in 7 per cent, with the contracting in 23 per cent.

2*d*, *Syphilis.*—This disease is universally recognised as a cause of waxy degeneration. In a very considerable proportion of the cases which I have observed during life, or examined after death, it unquestionably existed; in many of those which were complicated with tuberculosis it was present, but obvious grounds of uncertainty have prevented the accurate tabulation of the facts. From my notes, however, it appears that more than half of all my cases observed during life were so complicated.

3*d*, *Caries and Necrosis.*—These affections of bone have also long been recognised as causes of waxy degeneration, but they are much rarer causes than those already mentioned. Among my cases it appears that from 10 to 15 per cent may be reckoned as belonging to the group.

4*th*, *Chronic Suppuration* is believed by some to be the most important causal complication. It, of course, exists in all the cases of advanced tuberculosis, and of caries and necrosis. I have not seen suppuration independent of them lead to the disease, and in a considerable proportion of my cases it assuredly did not exist.

5*th*, *Cancer* is an occasional causal complication.

6*th*, *Chronic Rheumatism* is another disease which is occasionally known to precede, and perhaps to cause the renal affection.

Rosenstein* has collected from various sources a

* Op. Cit., p. 256.

series of 100 cases, among which the following were stated to be the causes :—

Tuberculosis pulmonum,	44
(Of these, ten cases were also affected with ostitis and one with syphilis.)	
Suppuration of bone,	29
Syphilis,	15
Empyæma,	3
Carcinoma,	3
Psoas abscess,	2
Pyelitis and hydronephrosis,	2
Abscess of liver,	1
Chronic alcoholism,	1

Of 20 cases recorded by the author * in 1861,

6 had phthisis.
6 ... syphilis.
2 ... caries.
2 ... general debility.
1 ... cancer.
1 ... chronic rheumatism.

My friend and former assistant, Dr Roberts Thomson † of Bournemouth, published an account of the causal complications which were found in a series of 50 cases examined post-mortem in the Royal Infirmary, Edinburgh. He found that—

Syphilis was noted in 12 cases, of which two were doubtful.	
Tubercle,	13 cases.
Diseased bone,	1 case.

* Edinburgh Medical Journal, February 1861. † Glasgow Medical Journal, 1866.

Thus the causal complication was recorded in only one-half of the cases, but of those which were recorded the proportion corresponded closely with my results.

B. Concomitant Complications.

The concomitant complications of waxy Bright's disease are similar degenerations of other organs and their consequences.

1*st, Waxy degeneration of the Liver* co-existed with the waxy kidney in about 70 or 80 per cent of all my Infirmary cases, and was never found in combination with the other forms of renal disease. It is often combined with fatty degeneration, and occurs in three forms—a diffused affection of the cells of the organ, an affection of the small vessels, and rarely in nodules here and there affecting all the elements. These conditions do not, so far as I am aware, lead to any important symptom, excepting the enlargement of the liver which accompanies them.

2*d, Waxy degeneration of the Spleen.*—The spleen was waxy in fully 80 per cent of my cases—the degeneration sometimes affecting the malpighian bodies and small vessels only, constituting the sago spleen, sometimes also affecting the pulp, leading to a general induration of the organ. In both of these forms the spleen is, as a rule, enlarged, and very commonly the blood shows, under the microscope, a morbid appearance. The number of the white corpuscles is increased, while the red are flabby, and have little tendency to form rouleaux, and, on the other hand, they tend to tail. I have found this condition in a large proportion of the cases examined during life.

3*d*, *Waxy degeneration of the Alimentary Tract.*—This degeneration affects the stomach and intestines; generally the small vessels are its chief seat, but it is not confined to them, for it occurs in the epithelial elements also. This complication was present in upwards of 50 per cent of my cases examined post-mortem. Very often during life symptoms referable to it occur. The degeneration of the stomach is accompanied by dyspepsia and occasional vomiting. The vomited matter is sometimes watery, sometimes bloody.*

The most prominent symptom of the degeneration of the intestinal mucous membrane is diarrhœa, which also is sometimes watery, sometimes bloody. But these symptoms are not constant, and I have seen some cases in which well marked degeneration existed without any diarrhœa.

C. Consequent Complications.

The consequent complications of the waxy kidney are not very important, but they essentially correspond with those of the other forms.

1*st*, *Affections of the Lungs and Bronchi.*—Congestion and œdema of these organs existed in 20 per cent of my cases,—a considerably smaller proportion than was found with the other renal diseases. Pneumonia also was rare, occurring only in 4 per cent. Moreover, it was specially frequent in cases fatal in the first stage. It seems to me that the co-existence was accidental.

2*d*, *Hypertrophy of the Heart* is a comparatively

* See British and Foreign Med. Chir. Review, Jan. 1868, p. 201.

rare result of this disease. I found it only in 4 per cent of my post-mortem cases, but all of these in which it occurred were cases in the third stage. It therefore appears to be a true consequence of the disease. Its occurrence is to be explained in the same way as in the inflammatory cases.

3*d*, *Inflammation of Serous Membranes* appears to be a rare complication of this disease. Pleurisy was present in 2 per cent; pericarditis in 8 per cent; and peritonitis in 2 per cent of my cases. The pericarditis, in particular, occurred more frequently in the advanced stages.

4*th*, *Diseases of the Nervous System.*—Serous apoplexy we pass over for the reasons previously mentioned. Sanguineous apoplexy occurred in 2 per cent of my fatal cases, and these cases were in the third stage of the disease.

5*th*, *Morbid Conditions of the Blood.*—In the course of this disease the blood becomes deteriorated, as is shown by the pallor which so generally attends upon its later stages. It appears to me that in three of the cases recorded by Dr Christison, and in which he analysed the blood, the waxy degeneration was the form of renal malady from which the patient suffered, although a certain amount of inflammation was in several superadded.

Case XIV.* was that of a medical practitioner in the north of Scotland, who died of intercurrent pleurisy. Some blood was drawn and examined. It presented a strong buffy coat, a scanty contracted crassamentum, and a very abundant slightly lactescent

* Op. cit., p. 227.

serum. The serum had a density of 1018·5, and contained only 6·16 per cent of solids. In 10,000 parts of blood there were only 491 parts of hæmatosin, 583 of solids of the serum, and 56 of fibrine. 500 grains of serum evaporated to dryness gave a fluid which yielded an abundance of pale pearly scales with nitric acid. In this case, from the absence of post-mortem examination, we cannot be sure of the lesion, but the symptoms are so characteristic that I feel satisfied of its waxy character. In commenting upon it, Dr Christison says, that the condition of the blood and the urine showed that the affection of the kidneys must have been very far advanced. The excessive reduction of the hæmatosin of the blood, namely to one-third of the healthy proportion, is best explained on the supposition of long continued and advanced disease of the kidneys. The abundance of albumen in the urine corresponds with its scantiness in the blood, which contained scarcely five-sevenths of the healthy quantity of that principle. The presence of a considerable quantity of urea in the blood is a somewhat anomalous fact, considering that the urine was secreted in large quantity.

In a similar case, No. XXV.* of Dr Christison's Appendix, he found the blood to have its colouring matter reduced to one-half the healthy proportion, while the albumen was diminished by nearly one-third.

In case XXVII.,† which is a more doubtful example of this disease, the blood was analysed and found to have lost one-half of its hæmatosin.

* Op. cit., p. 266. † Op. cit., p. 270.

If I be right in supposing that these were waxy cases, it would appear that the effect of the waxy form on the blood is very similar to that produced by chronic inflammatory changes,—viz., a diminution of the hæmatosin and the albumen, and a retention of urea.

CHAPTER XII.

THE WAXY OR AMYLOID FORM.

Treatment.

THE treatment of the waxy degeneration of the kidney is mainly the treatment of the constitutional state on which it depends or with which it is associated, and that consists of supporting and improving the general health. No medicine is known to produce a direct effect on the waxy degeneration itself. The constitutional debility requires tonic treatment; and considering the impoverished state of the blood, iron is manifestly the most suitable remedy of that class. It should be given perseveringly, the form being varied according to circumstances. The tincture of the perchloride with or without vegetable tonics, such as quassia or calumba, syrup of the iodide alone or in combination with cod liver oil, the compound syrup of the phosphates, and the saccharine carbonate, are the forms which seem most suitable. Other tonics, as quinine, nux vomica, and the mineral acids, are occasionally useful. The diet should be of the most nutritious description, but regulated with reference to the condition of the stomach. Porter, bitter ale, wine, and well diluted spirits are often useful, both as nutrients and stimulants.

The complications often require most careful man-

agement. *Tuberculosis* must be treated in accordance with the principles now generally adopted and well laid down in the standard works on medicine, and more particularly in books specially devoted to the subject, such as that of Professor Bennett. When the *syphilitic cachexia* is present, the patient is often greatly benefited by the use of iodide of potassium, given in doses of from two to five grains, and subsequently in larger amount if occasion require. I am assured by an able observer that he has found sarsaparilla also of great service. When *caries* and *necrosis* are causal complications, no effort should be spared to arrest the drain on the system by operative or other measures. The importance of the relation between these diseases of bone and the waxy degeneration should be carefully kept in view by surgeons. In *chronic suppurations*, also, we must strive to arrest the drain on the system, and towards accomplishing this end great benefit may be anticipated from the plan of treatment with carbolic acid introduced by Professor Lister.

It appears that none of the concomitant complications are amenable to treatment, except those of the *gastro-intestinal canal*, which manifest themselves by dyspepsia and diarrhœa, and even these too often defy our best efforts. At the head of the list of remedies available for the dyspepsia I would place nux vomica and its active principle strychnia. They may be given in the form of the extract of nux vomica, in doses of a quarter of a grain to two grains in pill, or of the tincture in doses of ten to thirty minims two or three times a-day, or, as I think, with better effect, in the form of the liquor

strychniæ in doses of five to ten minims several times daily. These remedies give tone to the stomach, and improve the digestive power. Mineral acids, particularly the dilute nitro-hydrochloric, may be given in the intervals between the doses of strychnia. Quinine also is sometimes undoubtedly beneficial, and, as has been already remarked, other bitter tonics may be given with advantage. *Diarrhœa* is, in many cases, the most troublesome complication. The ordinary astringents, such as catechu and kino, have not proved specially useful in my hands, and antacids, such as chalk, have been of little avail. Logwood is sometimes useful, but much less so than opium, which is, on the whole, the most satisfactory remedy. It may be given by the mouth, or in the form of enema or suppository. Astringents, such as gallic and tannic acids, may, with advantage, be combined with the opium, especially when it is given by the bowel. Lead and opium pills are also useful. In using opiates, we must keep in mind the tendency to head affections, and the danger of interfering with the renal secretion. Enemata containing solutions of nitrate of silver have repeatedly relieved patients in whom other remedies had failed. Solutions of other astringent mineral salts may be similarly useful. The dietitic treatment of this complication is also important. Finely ground farinaceous substances, such as corn flour and ground rice, serve to check the tendency to diarrhœa, whilst highly flavoured and spiced articles of diet produce the opposite effect. Plain roasted beef or mutton may be given, but soup and vegetables should be avoided.

The treatment of the consequent complications requires no comment beyond what was given under the inflammatory form.

A few words may be added with regard to the remedies chiefly employed in renal diseases. *Diuretics* are of little service in this disease,—the urine being already excessive, their action would be injurious rather than beneficial. They are, of course, useful when inflammation of the tubules supervenes. The most suitable forms are indicated in the chapter devoted to the combined waxy and inflammatory diseases. *Diaphoretics* might seem, at first sight, indicated by the dryness of the skin which sometimes attends the disease, but they are never really needed excepting when dropsy has appeared. *Cathartics* are seldom required, and should be used with caution, on account of the tendency to diarrhœa. I have seen them, however, eminently useful in cases in which inflammation of the tubules had supervened.

In this disease, also, change of climate is often of great advantage; and when this can be combined with the use of Chalybeate springs, considerable improvement may be anticipated.

While we cannot cure the disease, the question arises, Is it ever recovered from? No case is on record in which so happy an event occurred; but I have seen several in which the symptoms were well marked, and yet recovery took place. In one of the cases recorded I have sometimes hoped for a cure, the patient having repeatedly improved considerably, and being now in better health than eight years ago. In this case, that of Archibald March, recorded at page 85, there is

every reason to believe that the liver has improved, for it has diminished in size by several inches, and this has been accompanied by a constant improvement in his general health. It is quite consistent with what we know of pathology, that the waxy material might be absorbed and healthy tissue deposited in its place, but there is no evidence that this occurs.

Temporary improvement is very frequent; indeed, few patients fail to rally more or less under judicious medical treatment.

CHAPTER XIII.

THE CIRRHOTIC OR CONTRACTING FORM.

Morbid Anatomy.

THIS disease cannot, like those we have already considered, be said to pass through distinct stages, for it exhibits throughout its course one uniform character. *It consists essentially of an hypertrophy of the connective tissue of the organ, and a consequent atrophy of all the other structures.*

In the commencement of the process there is little diminution of the bulk of the organ, but the capsule is somewhat thickened, is less easily torn off than usual, and the surface, instead of being smooth, is uneven, scarred, rough, and granular. This is generally equally diffused throughout, but sometimes more marked at individual points. The colour is in some cases pale, in others reddish. Congested veins may often be seen ramifying on the surface. There is, in uncomplicated cases, little or none of the dense sebaceous-like material which is so constantly present in the atrophic stage of the inflammatory form. On section the cortical substance is found relatively diminished, the diminution most marked towards the surface. The small arteries are unduly prominent, their walls thickened, and their cavities often dilated. Even by the naked eye and by touch we recognise

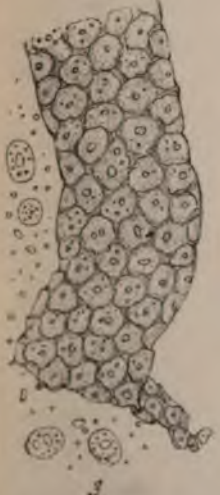
an increased density and fibrousness of structure. On the surface and in the substance, particularly the cortical part, cysts are frequently seen. On microscopic examination the one constant change is found to be increase of the fibrous stroma.

When the disease is more advanced the kidneys are much reduced in bulk, but the characters of the organ are essentially the same as those given above. The two kidneys may be of the same size, or one may be more atrophied than the other. It is, however, singular that even when the disease is furthest advanced the cones are comparatively little affected; the cortical substance may be reduced to a very thin layer, perhaps one-sixth of its natural size, and yet the cones be but slightly wasted. In this stage, also, it is remarkable how little sebaceous-looking material is observable in the tubes, but the prominence and thickening of the arteries is very marked. On microscopic examination with a low power (50 diameters) the small arteries are found much thickened, the malpighian bodies are closer together, their size varies more, and they are less conspicuous than they are in the atrophic stage of the waxy form; the tissue between them, particularly in the most atrophied parts, presents little or no appearance of tubules, it is an irregular but dense fibrous mass.—(*Plate* vi., *fig.* 1.) When the tubules are still present there is little of the opaque material which is so prevalent in the inflammatory cases, but it is not altogether wanting. Translucent hyaline matter is more common. Under a higher power (300 to 400 diameters) the increase of the fibrous tissue, and the atrophy of the tubular

PLATE VI.

1.

2.



3.

W.H. M.ª Farlane Lith.ª Edin.ª

elements, are very distinct. The malpighian bodies are seen to be surrounded by dense fibrous tissue, their capsules greatly thickened, and the tubules compressed and atrophied by the increase of inter-tubular substance.—(*Plate* vi., *fig.* 2.) At the same time many tubules may be found quite unaffected. Pigmentary matters are often seen in the tubes, principally within the epithelial cells.

Cysts are very common, especially in the cortical substance. Some originate in the capsules of the malpighian bodies, and the compressed group of vessels may be seen towards one side. Some result from obstruction of tubes and consequent dilatation of the distal portion. Some, again, are produced from morbid growth of epithelial elements.

A peculiarity occasionally met with, particularly in gouty subjects, deserves special notice. It was well described by Dr Garrod.* There is a deposit of a chalk-like substance in the form of streaks; these white lines are chiefly in the direction of the tubes, in the pyramidal portion; some, however, are in the cortical part. The mamilla of each cone also presents the appearance of little white points from the deposition of the same matter. These white deposits are composed of needle-like crystals of urate of soda, and are situated in the stroma of the organ, as well as in the tubules, chiefly in the former.

What is the true pathology of this disease? It has been very generally assumed that it results from inflammation of the stroma, in the course of which connective tissue is formed, and that this increased

* Garrod on Gout, p. 237.

formation leads to the secondary changes. There can be no doubt as to the latter part of the statement, but as to the former the evidence appears defective. I know of no observation which warrants the assertion, that in the earliest stage free exudation is to be found among the elements of the stroma, and, as we shall presently see, there is much reason to think that there is no such exudation. In the absence of direct evidence, this view appears to rest upon two considerations,—1*st*, that the formation of connective tissue is a common result of inflammation; and, 2*nd*, that the analogous disease of the liver is supposed to result from inflammatory action.

As to the first of these, it must be remembered that excessive formation of connective tissue is not exclusively connected with inflammation. It occurs as a distinct new formation in tumours, and unquestionably arises from simple hypertrophic growth. We are indebted to Dr Handfield Jones* for an elaborate and able discussion of the question of its non-inflammatory origin. After describing fibrous patches and deposits as they occur in the serous membranes, the arachnoid, the pericardium, the pleuræ, and the peritoneum, as well as in the valves of the heart, in certain forms of cirrhotic liver, in the mucous and submucous tissues of the stomach, in the substance of the testicles, the uterus, and the lungs, he argues: "that in all these instances the process may be from the first non-inflammatory, depending on the exuda-

* British and Foreign Medico-Chirurgical Review, 1854, Vols. xiii., p. 369, and xiv., pp. 36 and 329.

See Foerster's Handbuch der Pathologischen Anatomie, 2te Auflage, s. 1863.

tion of blastema, tending abnormally to fibre-development, and not simply maintaining the nutrition of the part." He says again, that the hypertrophy causing the increase and thickening of fibrous tissues, goes on and on, as a substantially independent process, totally unassociated with any trace of inflammation, even though it may by possibility have originated in it. My observations amply confirm the chief conclusions of Dr Handfield Jones. The only point on which I differ from him is, that I have not seen evidence of an exudation or blastema, but it is not improbable that in using these terms he was influenced by the pathological theories prevalent at the time he wrote.

From his observations, confirmed as they are by my own experience, I am satisfied that it is unwarrantable to assume, without positive evidence, that in any individual disease increased growth of connective tissue is a result of inflammation.

As to the second of these points, viz., the analogy of the cirrhotic kidney with the cirrhotic liver, it must be borne in mind, that in regard to the latter disease we have no positive evidence of inflammation. The stage of atrophy is the condition most commonly met with, and in it there is no free exudation among the connective tissue. The earlier stage in which the organ is enlarged comes occasionally under observation, but its increased size is found to depend solely upon connective tissue,—not upon free exudation. No observation is on record to show that a stage preceding this exists, in which free exudation is to be found. Thus the evidence with regard to the liver is

so unsatisfactory, that no argument from analogy can be founded upon it.

On the other hand, we have, I think, positive evidence that there is no free exudation in the cirrhotic kidney. We often have opportunity of examining cases which are evidently advancing, and in which no trace of such material can be found. Indeed, in every case the process is more advanced towards the surface than it is in the deeper parts, but here also a little examination enables us to make out that there is no evidence of exudation.

Moreover, in such an organ as the kidney, if free exudation be poured out, it must almost of necessity affect the tubules, and these are, as we have seen, not primarily involved. The clinical history of the disease also confirms this view, albuminuria not being an early symptom, which, in a truly inflammatory affection, it would certainly be.

It may be asserted that, though no free exudation exists, the connective tissue nevertheless results from an inflammatory process, but I know no evidence in favour of such a view, and the theory of a simple hypertrophy of connective tissue best explains the pathological and clinical phenomena.

The names which have been applied to this disease are very numerous. The "contracting kidney" is good, inasmuch as it refers to one of its most important and obvious features, but seeing that contraction or atrophy occurs in the advanced stages of the other forms of Bright's disease, the term is apt to lead to some confusion. The term "gouty kidney," although very appropriate in some cases, would be unsuitable in

many, inasmuch as, on the one hand, the disease often occurs independently of gout, and, on the other, gout may be accompanied by other renal diseases. The name "intertubular or interstitial nephritis" is also unsatisfactory, inasmuch as it assumes the existence of an inflammation of which, we believe, there is not sufficient evidence. The name "granular kidney" is inappropriate, inasmuch as the surface of kidneys, in the third stage of the inflammatory and the waxy disease, are often as distinctly granular as are those under consideration. The last name to which I shall refer, the "cirrhotic kidney," although etymologically incorrect, is, in my opinion, the most appropriate, as it indicates the most prominent feature and essential characteristic of the disease, viz., the increase of the connective tissue, and yet implies no theory as to its origin. For this reason I prefer to call it the cirrhotic kidney.

CHAPTER XIV.

THE CIRRHOTIC OR CONTRACTING FORM.

Clinical History.

THE earlier symptoms are very slight; such, indeed, as might easily escape notice. I have seen several cases in which the renal lesion was sufficiently distinct on post-mortem examination, but in which there had been no albuminuria, and no dropsy during life. Still, in a large proportion of the cases, symptoms do exist, and a careful observer will not fail to detect the malady.

It is specially a disease of male adults; occurring with increasing frequency in successive decades of life. It is more common in the gouty than in any other constitution, but is met with in all forms of the arthritic diathesis, and in many cases is unsuspected, until secondary diseases manifest themselves. I have at present under my care a gentleman, who considered himself in very fair health, until he was suddenly seized with an epileptic fit. On seeking an explanation of this attack, I found his urine distinctly albuminous, and obtained a history of this form of renal disease.

There is, in some cases, great thirst, and patients are obliged to rise several times every night in order to micturate. The secretion is often pale, and of rather low density. In amount it is at, or somewhat above,

the natural standard. These characters it generally retains throughout the whole course of the disease. In the early stages albumen is occasionally present, generally in very small quantity. One day it may be distinct, but the next day we search for it in vain. Tube-casts, hyaline or finely or coarsely granular, may also be found if carefully sought, but are very apt to be overlooked by the inexperienced observer. There is not unfrequently, even in the early stage, a peculiar anæmic appearance, and dyspepsia and thirst are much complained of. At first there is no dropsy, and cases sometimes run through their whole course without any distinct œdema appearing. But generally there is occasional swelling of the feet or ankles, and a degree of tightness of the boots is complained of at night, but it has disappeared in the morning. The eyelids are, however, often puffy, and the conjunctivæ dropsical, presenting the character styled by some physicians "the Bright eye." Ascites also is occasionally present, but the amount is trivial. In some cases, however, the dropsy is severe, and then it depends either upon an intercurrent inflammation of the kidneys, or upon cardiac, hepatic, or pulmonary complications. As the malady advances the general health gives way, the strength diminishes, the anæmia becomes more pronounced. There is an increasing unfitness for exertion. The patient has a chronic cough, has little power of reaction, easily catches cold, and often suffers from coronal headache. In this state he may linger for months, or it may be years. But the disease is making progress: gradually the albumen increases in the urine, the appearance grows

more unhealthy, certain pulmonary and gastric symptoms become distressing, the strength diminishes, and gradually or suddenly a fatal aggravation of the symptoms ensues. He is seized with uræmia, with apoplexy, with sudden œdema of the lungs, or with acute inflammation, and so dies. A patient, originally of vigorous constitution, sometimes survives the most alarming attacks, and is restored for a longer or shorter time to comparative health; but at length they recur, and he sinks often suddenly in the end. The variety in the nervous symptoms in this disease is very remarkable; convulsions, coma, delirium, delusions of the senses, and maniacal excitement, are each of them occasionally met with.

In illustration of the clinical history, I select the following cases:—

CASE XXV.—*Cirrhotic kidney, tubercle of lungs and intestines; no dropsy nor albuminuria.*—J. M—, æt. 65, was a gardener, and was admitted to the Royal Infirmary, under the care of Dr Sanders, November 7th, 1865. He had always been a sober and generally a healthy man till the end of September, when he took cough and pain in the chest. The sputum was purulent. The urine was 40 oz. daily, acid, of sp. gr. 1020, contained no albumen nor any deposit. He had no dropsy. Towards the end of November he took diarrhœa, and from that time became worse. He died January 20th, 1866.

Autopsy.—The body was emaciated. The visceral layer of the pericardium was œdematous. The heart substance was fatty; the valves were competent, but somewhat thickened; the aorta was atheromatous. Both lungs contained tubercle and tubercular cavities. The liver was fatty. The spleen was natural. There were some tubercular deposits and ulcers towards the lower end of the

ileum. The kidneys were small and pale, their surface slightly granular; the cortical substance was somewhat diminished; the fibrous stroma was distinctly increased. A few of the tubules contained exudation and some a little pigment, but the majority were quite natural.

Commentary.—In this case we had a distinct change in the structure of the kidneys without any urinary symptom, the quantity and quality of the fluid having been natural. The death resulted from the tubercular disease of the lungs and intestine.

CASE XXVI.—*Cirrhotic kidney, dilatation of heart, emphysema of lungs, general dropsy, albuminuria.*—T. R—, æt. 67, had been admitted to the Royal Infirmary, under the care of Dr Sanders, with dilatation of heart, bronchitis and emphysema, albuminuria and general dropsy. These symptoms gradually increased, and proved fatal a few days after admission.

Autopsy.—The heart was enlarged, particularly the left side; the right cavities were dilated. The aorta was somewhat atheromatous, the valves were competent. The lungs were congested, their lower part almost pneumonic; they contained some patches of pulmonary apoplexy. The liver was congested and soft. The spleen was firm. The kidneys were congested, their surface granular, the cortical substance relatively diminished; many of the tubules contained exudation, others were natural; the connective tissue was increased. The intestines were natural.

Commentary.—This case illustrates the fact that cirrhosis of the kidney is often associated with cardiac and pulmonary disease, and that when this is the case, the symptoms are much more pronounced than in the uncomplicated cases. It was scarcely an example of pure cirrhosis, for a considerable amount of exudation occupied many of the tubules. Still, the

cirrhosis was quite distinct, and was not, in my opinion, a result of the cardiac affection.

CASE XXVII.—*Cirrhotic kidneys, polyuria, bronchitis, pleurisy, and pericarditis, uræmia, death.*—John O'Hara, æt. 23, was admitted into Ward VII. under the care of Dr Balfour, on October 26th, 1867.—States that he has only been ill four weeks, that he has always previously been healthy, and, in particular, that he never suffered from syphilis. From his known dissipated character, all these statements are not, however, to be implicitly trusted.

On admission.—He suffers from debility, with considerable thirst, attended with the passage of a pale, almost colourless albuminous urine, averaging above 120 oz. in the twenty-four hours. The albumen ranges from a mere trace when passing only 100 oz. of a density of 1012, to $\frac{1}{10}$ when passing 125 oz. of a density of 1011. The average specific gravity was 1011. There are occasional hyaline casts observed. He has fatty degeneration of both retinæ, as observed through the ophthalmoscope. During his residence in the ward, he had occasional attacks of hemoptysis, and from this and the existence of dullness beneath the right clavicle and slightly prolonged expiration, the existence of tubercle in that part of the lung was inferred. He was treated with full doses of iodine, with the view of lessening his thirst and polyuria, without effect. Subsequently full doses of the perchloride of iron were administered, and for a time his drink was restricted, but no amount of restriction sufficed to reduce his urine below 80 oz.—and the actual amount of his polyuria may be inferred when it is mentioned that this restricted amount, which lasted for a few days, is included in the average given above. He was discharged on the 22d of January 1868, in *statu quo*.

On the 15th of April he was re-admitted into Ward IX., in a semi-unconscious state from drink—covered with bruises, and labouring under severe bronchitis. From his noisy behaviour he had to be removed to Ward X., and after some days' treatment—chiefly with carbonate of ammonia—being apparently somewhat

quieter, he was again removed to Ward VII. He never properly recovered consciousness, was always more or less noisy, complained of no pain, and nothing could be detected in his chest but loud sonorous rales and coarse crepitation, while his amaurotic condition precluded any diagnostic value being attached to his dilated pupils. He died on the 25th of April—noisy to the last.

Autopsy.—The face and feet were slightly œdematous. There was recent pleurisy of the left side. The lungs were emphysematous at their margins, and œdematous throughout. At the apex of the right, there were traces of old tubercle, and in the middle lobe there was a considerable amount of recent tubercular deposit. The pericardium (visceral and parietal) was coated with a thin layer of recent lymph; the membrane was congested. The heart was dilated and hypertrophied; its valves were natural; its auriculo-ventricular orifices dilated; its muscular substance was granular and fatty. The peritoneal cavity contained a considerable amount of yellow serum. The liver was enlarged, somewhat congested. There was some increase of connective tissue towards the centres of its lobules, round the hepatic venous radicles. The spleen was normal. The kidneys were much diminished in size; their capsules were somewhat adherent; the surface rough and granular; the cortical substance was greatly reduced in size relatively to the cones. On microscopic examination, the fibrous stroma was found much increased; the vessels were not waxy, but the smaller arteries were thickened; many tubules were completely atrophied, and some contained cells in a state of fatty degeneration. The brain was natural.

Commentary.—This patient was a man of dissipated habits, and never had nephritis. The renal disease must have been of long standing, although he dated its commencement only six months before his death. Dr Balfour was inclined to suspect waxy disease, from the large amount of water passed, but the post-mortem examination proved the case to be one simply of the cirrhotic form. It is thus interesting, as showing that polyuria, although so constant in the waxy

disease, is not exclusively associated with it. The recent pericarditis and pleurisy, as well as the fatty degeneration of the retina, and the consequent amaurotic condition, were well worthy of attention. It may be doubted whether the nervous symptoms which he exhibited after his return to the Hospital were really uræmic, but considering that they lasted for ten days after the debauch which induced the fatal attack, we are warranted in believing that they were. There was no convulsion, simply a drowsy comatose condition.

In recording the next case, I cannot do better than extract a portion of its history from Dr Bennett's work * on the Principles and Practice of Medicine.

Case XXVIII.—*Chronic gout, with tophaceous deposits in all the joints, cirrhotic kidneys, pneumonia, pyæmia, (?) death.*—Thomas Burns, aet. 42, a tobacco-pipe maker, admitted November 4th, 1857. Says he first became ill in Glasgow about ten years and a-half ago, with pain and swelling in both his big toes. Soon afterwards the ankles and knees became affected. He was confined for a month, being unable to walk, or even to put on his shoes. Since then he has had on an average three such attacks every year, spring and autumn being the worst seasons; but he has rarely been confined by them more than a week. The attacks have generally commenced with rigors, followed by more or less fever and swelling in one or other of the joints. Almost every joint in his body has suffered in this way at one time or another. At the first attack, he says, chalk stones formed in his toes, and since then they have appeared in his feet, knees, elbows and hands. The right hand especially has been much deformed by them. He is in the habit of cutting down upon, and extracting them, whenever they approach the surface and are unusually painful. He has been twice in the Infirmary, and on both occasions dismissed relieved. The present illness commenced sud-

* The Principles and Practice of Medicine, by John Hughes Bennett, 5th edition, p. 991.

denly six weeks ago, and has more especially affected the ankles. He has undergone a great amount of treatment, having been bled and cupped, and having taken much medicine. He had been accustomed to drink a good deal of porter, as well as of spirits, until three weeks before his first admission, in June 1856, since which time he has been more temperate.

On admission.—He complains of pain in the left wrist and both ankle joints, which latter are swollen, and pit on pressure. The joints of the fingers are nodulated and crooked, especially those of the right hand, hard to the feel, with numerous tophaceous deposits visible through the shining and stretched integument, about the size of millet seeds. The elbow and knee joints are similarly affected with several deposits over the olecranon and patella of each limb. The toes are not so distorted as the hands. There is a pain on pressure over the right lumbar region, with a slight trace of albumen in the urine. Other functions normal.

November 25th.—Small abscesses have appeared over the patella and heel, to which poultices have been applied. . . . . .

Dec. 18th.—Last night was seized with severe lumbar pain, and general febrile symptoms, and on examining the urine it was found to be highly albuminous. The sediment contained numerous epithelial cells from the kidney, with granular and desquamative casts of the tubes.

Dec. 21st.—Is much better. Albumen in the urine diminished.

Jan. 6th, 1858.—Since last report has been comparatively free of pain, and doing well, but last night was again seized with severe febrile symptoms, accompanied by painful sensations throughout his body. To-day the joints of the extremities, especially those of the hands, are very painful.

Jan. 8th.—He has been perspiring much, and is better, although pains in joints are still very severe. The poultices have brought away several fragments of the tophi near the surface. They are of a pale yellow colour, friable, and when examined under the microscope present a mass of needle-shaped crystals of urate of soda.

Jan. 22d.—The pains in the joints have now been absent for ten days, and he was dismissed.

Thus far I have quoted from Dr Bennett, omitting only the various prescriptions which were given during the prolonged residence in the Hospital.

He had no attack of gout from the time of leaving the Infirmary until early in January 1860, when he was seized with rigors, which were followed by deposit of soft pultaceous fluid in the neighbourhood of the joints. These deposits gradually dried up. The joints of the hands and feet and the knees were affected. The heart sounds were natural. There were signs of some consolidation towards the upper parts of both lungs. There was no pain over the kidneys. The urine was in good quantity, of a pale amber colour, specific gravity 1011, contained much albumen, and a few hyaline tube-casts. He slept badly on account of the constant pain in his feet. His tongue was furred and dry, his appetite poor, and his bowels loose.

April 21st.—Since last report, the patient has continued in the Hospital, and has had occasional acute exacerbations of pain, generally attended by deposits of urate of soda in the joints. The successive deposits were at first fluid, but gradually dried up and formed chalk stones. The urine varied from 70 to 90 oz. daily, but never was in excess of the fluid consumed. It was always albuminous, was sometimes loaded with urates and sometimes with phosphates, and on careful examination a few hyaline casts could be detected.

June 9th.—After the date of last report the patient gradually improved for some weeks; but about the end of May he was seized with subacute pneumonia, the amount of urine then diminished to 30, 20, and ultimately to 12 oz. per diem. He had no dropsy. He never had any convulsion nor loss of consciousness, but became quite insensible to pain, and assured me almost with his last breath that he was feeling better. He died on June 9th.

Autopsy.—Heart and pericardium natural. There were old adhesions on the right side of the chest, recent lymph and a little turbid fluid were found in the left side. The lungs were dark coloured, and in a state of solid œdema, each contained a little tubercle in the apex. Abdomen—On cutting into the liver, several small abscesses were found scattered through it; each

contained a little pus. There was no increased vascularity around them. The left kidney was very small, and irregular on the surface. On cutting into it a number of small yellowish white deposits of urates were seen in the medullary substance. The right kidney, though small, was larger than the left; it felt firm, was somewhat irregular on the surface, and contained two or three cysts, and several small deposits of urates. In the spleen several small abscesses, similar to those met with in the liver, were found.

Commentary.—This patient was under the care of Drs Bennett and Laycock successively, and was for a considerable time well-known to the students attending the clinical wards. He exhibited a typical example of gout and of the gouty kidney. The urine was in good quantity, but never in great excess, its specific gravity was rather low, and it generally contained albumen and tube-casts, though not in large amount. He never had dropsy. The state of the nervous system for some days before death was very peculiar. There were no convulsions, scarcely even a twitching of the facial muscles. The consciousness of mental impressions remained perfect to the last, but there was no sense of pain or weariness.

CASE XXIX.—*Cirrhotic kidney, slight albuminuria, dropsy.*—S. M., a house-painter, æt. 53, resident in Edinburgh, was admitted to the Royal Infirmary under my care, April 24th, 1866. Patient stated that as a rule his health had been good till 1863, but within the three years intervening he had had five attacks of lead colic. Being a painter by trade, he was much exposed to the lead poisoning. Three years before he found his health failing, he was easily fatigued, and his breath was very short.

On admission.—He was found to be labouring under chronic pleurisy and pericarditis, with little effusion. He was passing

urine in fair quantity (45 oz. per diem.) It contained albumen in small quantity, and was of fair specific gravity. He was very anæmic and feeble, had slight dropsical effusion in the eyelids and feet. The liver and spleen were natural. The extensors of the forearm were feeble and wasted, but there was no actual paralysis. He was treated with chalybeate tonics and diuretics, and after a few weeks' treatment so far improved as to be able to leave the Hospital. Some months afterwards he returned, presenting much the same symptoms, but considerably weaker than he had formerly been. Under treatment he again rallied, and I have not seen him since.

Commentary.—This case, although incomplete in consequence of my having lost sight of the patient, was undoubtedly one of the cirrhotic kidney, and it illustrates several points in the natural history of the disease. The patient was a house painter, and he had suffered from lead poisoning, a condition which, as we shall presently see, occasionally causes the disease. The quantity of urine was about the normal; and it was but slightly albuminous. There was some dropsy and considerable anæmia. The dropsy yielded to treatment, and the blood also materially improved under the remedies employed.

CASE XXX.—*Gout, albuminuria, cirrhotic kidney.*—John Stewart, a native of Manchester, a coppersmith, æt. 43. Came to Edinburgh for medical advice, and was admitted to the Royal Infirmary under my care in March 1868. Patient stated that he enjoyed good health up to the age of 14, when he had an attack of quinsy, which terminated by bursting and discharging matter. This quinsy returned regularly every year up to the age of 24. At 25 he went to America, and after four or five years' residence there he was attacked by ague. This kept him laid up for about two years, when, thinking there was no chance of his getting better if he stayed in America, he returned home, and

soon afterwards became quite well. He was treated with quinine in America, but he thought with little benefit. About six years after the disappearance of the ague he was attacked by gout. It began by a pain in the ball of the great toe of the left foot, coming on suddenly about two or three o'clock in the morning. By the next day the pain had extended to the ankle, where it continued for three days, and then disappeared. Three weeks afterwards pain appeared in his left elbow, and continued shifting about till two years ago, when he was attacked by rheumatic fever. Since that time the pains have never left him. Towards the end of the fever, a pain came on in the cardiac region. This has been gradually getting worse, and on the slightest exertion he was subject to breathlessness and violent beating of the heart. The day before he entered the hospital a severe pain came on in his left wrist. His father, he states, died of a stomach complaint, probably gastric ulcer. His mother is living, but subject to rheumatism. His sisters are healthy. About the time when he went to America, he first began to drink ale and porter, and has been a beer drinker to a considerable extent ever since.

On admission.—Respiratory system normal. Circulatory system normal. Digestive system—Tongue dry, and covered with a whitish brown fur. His appetite is not good, but what food he takes agrees with him pretty well. He complains of great thirst. (This thirst continued up to the day of his death, and during all his stay in the ward he drank large quantities of water, lemonade, &c.) His bowels are regular. The spleen is considerably enlarged. Nervous system—Complains of pain in the left hand and wrist, which are swollen, and tender to the touch. Integumentary system—Perspires a great deal at night. Genito-urinary system—Urine has an acid reaction. Sp. gr. 1012, contains a small quantity of albumen. He was treated with acetate of potass and actea racemosa with little benefit.

April 7th.—Symptoms of pericarditis made their appearance.

April 12th.—Patient complains of a superficial ulceration on the mucous member of his mouth.

April 15th.—Ordered to have liquor epispasticus applied to his left wrist, and the effused serum to be kept and tested for uric acid. The serum effused from the blister was tested, but no

uric acid could be discovered. The pericarditis never became severe, and gradually subsided.

May 10th.—The pains have been shifting about from joint to joint a great deal for some days past, but have begun to disappear. Urine still continues albuminous. Quantity passed 108 oz.

May 12th.—The urine was analysed. Quantity of urine passed was 100 oz., and the amount of urea was 40·64 grammes = 627·23776 grains, or 1·31 oz. in 100 oz.

20th.—The patient has so far improved as to be able to leave the Hospital.

Commentary.—This was a typical case of cirrhotic kidney, as was afterwards proved by post-mortem examination. The previous history and the symptoms were alike characteristic of the disease. The further history of the case is given as illustrating the combined cirrhotic and inflammatory diseases, of which the patient ultimately died. I shall refer to one point only at present, the analysis of the urine. The amount of urea was not diminished; it was, indeed, nearly as high as the maximum of the tables given by Dr Parkes.* This was very interesting, considering that the kidneys were considerably wasted.

* The Composition of the Urine, by Edmund A. Parkes, M.D., p. 7.

CHAPTER XV.

THE CIRRHOTIC OR CONTRACTING FORM.

Nature of the Symptoms.

In this disease, again, we have to consider the symptoms connected with the urine, the dropsy, and those dependent on the nervous system.

I. *The Urine.*—The quantity is, in the earlier stages, at or somewhat above the healthy standard, and even when the kidneys have become considerably wasted there is no change in this respect. The tubules are not occluded, and so the watery elements which are separated from the blood flow unobstructed outwards. But how does it happen that while much of the kidney is atrophied, a natural or even excessive amount of fluid transudes? It may perhaps be explained by the fact that increased blood pressure is exerted on the malpighian bodies which are still in action; but such an explanation is unsatisfactory, inasmuch as though it might account for the urine not being diminished, it cannot account for the actual increase. There may be some change in the capillary walls, whereby excessive transudation may occur, but such a change has not yet been discovered. It is not unlikely that in many cases the amount of urine is regulated by the amount of fluid consumed, but this

cannot be accepted as the sole explanation, seeing that in one of the cases recorded experiments were carefully conducted, from which it appeared that diminution of supply of fluid did not correspondingly influence the secretion of urine. Perhaps the true cause may one day be found in some abnormal condition of the blood, whereby its watery parts more readily permeate the capillary membrane. Albumen is rarely present in any considerable quantity, and its presence—fitful in its appearance, and varying in its amount—is equally difficult of explanation. The tube-casts are generally few in number, hyaline or finely granular, but sometimes fatty. They consist of coagulated fibrine which has been effused into the tubules, mingled with more or less altered epithelium. The specific gravity is commonly low, and the colour pale. I have found in one or two well marked cases the quantity of urea little if at all below the natural standard. Other observers have found the urea distinctly decreased, particularly in the later stages. Uric acid is also reduced or quite absent in advanced cases. Phosphates, sulphates, and chlorides are also much diminished.

II. *Dropsy* is in some cases entirely absent, but more commonly is occasionally present, especially as the disease approaches its fatal termination. Even then it has, in the cases which I have observed, appeared only when an inflammatory exudation into the tubules had become superadded to the primary disease. In several well-marked cases, such as that of Thomas Burns, I have seen the patient free from dropsy to the last. The slight degree of œdema which

frequently occurs may be due either to the anæmia or to the renal affection, or to their combined influence.

III. *The nervous symptoms* commonly included under the term uræmia, not unfrequently make their appearance in this disease when the case is far advanced and still uncomplicated, but more particularly when an inflammatory condition has been superadded. The symptoms are very various, sometimes convulsions, sometimes delirium, and sometimes coma being most prominent. Moreover, they come and go in a singular manner, being at one time severe, at another time slight—appearing and disappearing in a way that we cannot explain. The mode of production is probably here essentially the same as in the other forms—much of the renal secreting structures being destroyed, a retention of the excrementitious matters, which are usually eliminated by these channels, necessarily follows, and it is to this retained material that the symptom is due. But in the uræmic attacks of cases of this kind, I have frequently been struck with the anæmic appearance of the patient, and have been led to suspect that the deficient nutrition of the brain had induced the attack.

Another nervous symptom which is worthy of special attention is headache ; the crown of the head is its special seat. It resembles in character that which sometimes follows severe hemorrages. It may exist for long independently of uræmia, and seems to arise from the deteriorated state of the blood. Certainly its amenability to treatment by means of the chalybeate tonics favours this view.

Yet another nervous symptom often occurs, viz.,

pains of a rheumatic character; at all events, pains which are generally described by patients as rheumatic. They flit about from part to part, but tend specially to affect the extremities, and are generally difficult of cure. These symptoms, though most markedly connected with the cirrhotic affections, do sometimes co-exist with the other forms.

CHAPTER XVI.

THE CIRRHOTIC OR CONTRACTING FORM.

Complications.

In considering the complications of the cirrhotic kidney, we shall take first those of the consequent, and second, those of the concomitant class. Both are of much importance in relation to the natural history of the disease. It is indeed not uncommon for the renal malady to be detected only when the complications have drawn attention to it.

A. Consequent Complications.

1*st*, *Hypertrophy of the Heart* is of frequent occurrence, being sometimes a result of concomitant valvular or vascular diseases, sometimes a result of the renal malady alone. In a series of cases which I examined post-mortem, nearly one-half (46 per cent) had enlarged heart, simply from kidney disease, while many others had enlargement connected with the lesions above referred to. I believe that it is present in almost every advanced case, though less common in the early stages.

It owes its origin in this affection to the same circumstances as in the diseases already considered, viz., impurity of the blood from imperfect elimination. The obstruction to the circulation in the kidneys

which arises during the progress of the disease may also, in some degree, contribute to the result. I have rarely heard patients complain of uneasiness from cardiac hypertrophy, but have often found it distinctly evidenced by physical signs during life.

2*d*, *Affections of the Lungs and Bronchi*:—

a. *Congestion and Œdema* are very common in this disease. In more than half of my cases they were well marked. Sometimes coming on very suddenly, they simulate an attack of acute bronchitis. Whether coming on suddenly or insiduously, they are frequently the cause of death.

b. *Pneumonia* was present in 7 per cent of my cases, and in these it no doubt contributed to, if it did not actually induce, the fatal result.

3*d*, *Inflammation of Serous Membrane*.—Pleurisy was observed as a recent lesion at the time of death in 15 per cent of my cases, pericarditis in 7 per cent, peritonitis in none. In the cases in which this affection was present the renal malady was generally well advanced, and it appeared that the complication had been the cause of death. With regard to the probable mode of origin of these inflammations, it is unnecessary to add anything to what has already been said.

4*th*, *Derangements of the Alimentary Tract*.—In some cases post-mortem examination reveals signs of chronic inflammation of the gastric mucous membrane, affecting both the tubes and the intertubular substance. Dr Fenwick* remarks that the granular kidney and other chronic forms of Bright's disease are most associated with intertubular gastritis, while the

* The Morbid States of the Stomach and Duodenum, page 178.

acute renal affections are accompanied by disease of the follicles. Dr Wilson Fox confirms these observations. Dyspeptic symptoms are commonly urgent and distressing. Thirst, feeble digestion, acidity, and flatulence are often complained of by patients.

5*th, Diseases of the Brain.*—Sanguineous apoplexy is a more common cause of death in this than in any other renal malady. It was present in 15 per cent of my series of cases, and in most of them it had led to the fatal result. Most common in the advanced stages, it may be due in part to the altered state of the blood, in part to the increased force of the hypertrophied heart, and in part to the degeneration of the vessels which so frequently accompanies the disease.

6*th, Affections of the Eyes* have long been recognised as occasional complications of albuminuria. They may be met with along with other renal affections, but I have hitherto seen them only with cirrhosis. Dr Badar's* experience evidently coincides with mine, for he says that "granular kidney, with dilatation of the cavities of the heart and hypertrophy of the left ventricle, have been found not in all but in most cases in which post-mortem examination could be obtained." And, in another place, he remarks that the renal symptoms preceding the ophthalmic are often so slow in progress and slight in degree as to escape notice. There are two morbid conditions of the eye which deserve attention.

a. Uræmic Affection is comparatively rare. (Dr Badar observed it in 6 out of 38 cases.) The patient

* The Natural and Morbid Changes of the Human Eye, by Charles Badar, Ophthalmic Assistant-Surgeon to Guy's Hospital, 1868, p. 464.

becomes suddenly blind; remains so for a few minutes or hours, and then quickly recovers. Vision, though unaffected after the first attack, fails gradually when other nervous symptoms have been superadded.

b. Retinitis and its consequences.—The impairment of vision caused in this way is less sudden, but progresses gradually and varies in degree. Its occurrence is at once apparent, because the region of the yellow spot is generally affected, and thus the patients complain of a mist obscuring their vision, and objects are seen most distinctly when held to one side. As in other forms of retinitis the ophthalmoscope reveals congestion, swelling, and loss of transparency of the retina, but, in addition, there are certain characteristic appearances,—brilliant yellowish white and opaque spots round the optic disc and in the region of the yellow spot, and numerous minute extravasations into the substance of the retina. The yellow patches are held by some to result from transformation of extravasated blood. In earlier stages, indeed, the red clot may be discerned, and not uncommonly we find red and yellow patches side by side. In many cases the retinal substance between the spots is restored to a healthy state while the patches continue. Some observers conceive that the patches are not entirely due to extravasated blood, but depend upon simple degeneration of the retina. I know of no theory which satisfactorily explains these retinal affections.

7th, Affections of the Blood.—In the more advanced stages of the disease there is obvious anæmia, and a condition corresponding to that described as occurring in the renal affections already considered.

B. Concomitant Complications.

1*st*, *Affections of the Liver.*—The most common change is cirrhosis, which may be more or less marked. It was present in 15 per cent of my cases. Fatty degeneration occurs in nearly the same proportion.

2*d*, *Affections of the Spleen.*—Thickening of the capsule and increase of the fibrous stroma commonly co-exist with cirrhosis of the kidney. They were present in about 40 per cent of my cases.

3*d*, *Affections of the Blood Vessels.*—Atheroma of the aorta and the systemic arteries generally occurs very commonly as a concomitant complication. We find it in all its stages, from the mere sclerosis of the inner coat to fatty disintegration and calcareous impregnation.

Tubercle of the Lungs is often met with in cases of this form of Bright's disease, but phthisis is so common in this country that it is difficult to determine whether it is merely a casual complication or in some way related to the renal malady.

CHAPTER XVII.

THE CIRRHOTIC OR CONTRACTING FORM.

Causes.

THE name gouty kidney is a sufficient indication of the frequent co-existence of this disease with gout. Dr Todd,* who published an able lecture on the subject, remarks that, while this malady may occur in other states of the system, it is peculiarly apt to be developed in the inveterate gouty diathesis. He describes several well-marked cases, and points out how well their history comports with the humoral view of the pathology of gout. Dr Garrod also describes cases of this kind in his work on gout. I have frequently met with cases in which these maladies co-existed, but have also not unfrequently seen the renal disease, independent of gout. On the other hand, I have seen one and known of other well-marked cases of rheumatic gout in which the kidneys were waxy; and again, I have seen gout accompanied by inflammatory Bright's disease.

With regard to the mode of action of the gouty poison in inducing this state of the kidney, Dr Todd remarks that such a condition as this may be easily produced by a tainted nutrition. The blood, charged with the morbid matter of gout, furnishes to the

* Clinical Lectures on certain Diseases of the Urinary Organs and on Dropsies, by Robert Bentley Todd, M.D., page 313.

kidneys an unhealthy pabulum, which, while it undergoes changes analagous to those which occur in health, does so in a very imperfect way, insufficient to maintain the nutrition of the healthy tissues of the gland. This, he thinks, explains the shrinking of the organ; the altered circulation would lead to the albumen in the urine, the want of epithelium to its pale colour and low specific gravity. This theory, although interesting, can scarcely be accepted as a solution of the question.

We are indebted to Dr Garrod for the observation that the *introduction of lead into the system* favours the occurrence of gout and of the cirrhotic kidney.* His observations have been amply confirmed by other observers, among whom may be mentioned Dr Warburton Begbie† and M. Charcot.‡

M. Ollivier§ found, in a series of cases occurring in workers in lead, who were neither addicted to drinking nor cachectic, that albumen was present in the urine. Sometimes it was merely temporary, sometimes more persistent. When temporary he conceived that it resulted from the irritation of the lead passing through the kidneys. When permanent he believes it to result from degeneration of the renal tissues consequent upon the deposition of lead within them.

Lancereaux‖ found, in several cases which he examined, granular kidney. I have carefully observed several cases in which Bright's disease co-existed with lead poisoning, or occurred in workers in lead, and have found that this form of renal disease was the one from which they suffered.

* Garrod on Gout. † Edinburgh Medical Journal, August 1862.
‡ Gazette Hebdomadaire, 1863, No. 10 et 27.
§ Archives Générales de Médecine, 1863-4. ‖ L'Union Médicale, 1863.

Some writers are of opinion that *chronic congestion of the kidney from heart disease* leads to this form of renal affection. It is quite true, as Sir William Jenner* pointed out, that chronic congestions lead to induration of organs. Such lesions may be constantly observed in the spleen and liver. I have also seen, as the effect of the same causes, induration, and less frequently a granular condition of the kidney. But the tubules were affected as much as, or even more than, the fibrous stroma, the very reverse of what we see in true cirrhotic disease. In the earlier stages of cardiac disease albumen not unfrequently appears in the urine, the result of increased blood pressure on the capillary walls; and, as a farther consequence, the tubules are blocked up with a fibrinous exudation. Some of this is discharged in the form of fibrinous casts, but a portion remains, and being gradually absorbed, leads to atrophy, corresponding to the amount of renal structure destroyed. When the congestion has existed long, the kidney undergoes induration, but the process appears to me to correspond to what is termed the spurious cirrhosis of the liver, which arises under similar circumstances.

Pregnancy has also been supposed by some authorities to lead to this affection, but I have not met with any cases confirmatory of the view.

Drunkenness is, in my experience, a very common characteristic of the victims of this form of renal disease, and although I have not been able to obtain statistical evidence on the point, I am satisfied that a connection really exists between cirrhosis of the kidneys and intemperate habits.

* Medico-Chirurgical Transactions, Vol. lxiii.

CHAPTER XVIII.

THE CIRRHOTIC OR CONTRACTING FORM.

Treatment.

THIS is the most hopeless of all the forms of Bright's disease in relation to treatment, for we have no means of curing the renal disease, and it is even doubtful whether we can check its progress. The remedies available for the alleviation of symptoms are also uncertain in their action.

But while this is true, the physician can do much for his patient by removing, curing, or diminishing the conditions which are known to cause and aggravate the disease, by relieving the symptoms which arise in its course, and by obviating or treating the complications as they occur.

Among the causes, we have already seen that *gout* is entitled to occupy a prominent position, and in many cases its treatment is as important as that of the renal affection itself. The patient must be encouraged to take sufficient out-door exercise, but exercise short of fatigue. Riding may be specially recommended to those whose circumstances admit of it. The patient must also be careful to avoid excessive mental work; and this can best be managed in the case of busy men by sending them to watering-places, or to travel. The diet must be nourishing, but not

stimulating, containing a fair quantity but not an excess of animal food, and a considerable proportion of vegetables. With regard to wines, claret and hock should be preferred, while the heavy, the sweet, and the sparkling varieties should be avoided. If stimulants be required, brandy or other spirit may be given, but should be well diluted, and partaken of sparingly. Potash or lithia water may be taken with advantage; soda is less suitable, for uric acid, which is in excess in the blood, forms with potash a soluble, with soda an insoluble salt. Of medicinal remedies, the most valuable are colchicum and alkalies, such as potash and magnesia. In gouty cases, tonics are frequently indicated, and much benefit may be derived from judicious hydropathic treatment.

When the renal affection depends upon the presence of *lead in the system*, our first aim must be to prevent further contamination. In the case of tradesmen, such as house-painters and plumbers, it is difficult to effect this, but we can warn them to be cleanly, to avoid eating in the midst of their work, and to keep away from those particular departments of work which are specially dangerous. We must further attend to the state of the bowels, and endeavour, by medicines, to prevent the absorption of lead, to get it out of the system, or to render it inert. Iodide of potassium may be given with a view to the formation of iodide of lead. Sulphuric acid also may be employed in order to produce the inert sulphate of lead. It may be given in the form of acidulated lemonade, or simply with water.

In the treatment of the symptoms directly referable

to the renal disease, little can be done to improve the condition of the urine. If its quantity become diminished, we obtain good results from diuretics. If the action of the skin be defective, diaphoretics and baths may be tried. If dropsy occur, it must be treated on the principles formerly explained, regard being always had to the impoverished state of the blood. If uræmic convulsions occur, bleeding or chloroform may be resorted to; the anæmia renders the latter agent preferable. Even in this condition tonics may be useful. The coronal headache frequently yields to chalybeate tonics, which are also sometimes useful for the rheumatic pains in the limbs.

With regard to the complications, the pulmonary œdema and the acute inflammations should be treated on the principles previously enunciated, but special caution must be exercised as to the employment of depletion, mercurials, or other lowering remedies.

The affections of the primæ viæ also frequently require patient management. When sickness and vomiting occur, ice and other gastric sedatives are useful. Strychnia and nux vomica are very useful as tonics; other bitters, with or without iron, are frequently most valuable.

When the eyes are affected, all exertion and exposure to strong light must be avoided. The patient must not read, nor attempt to do any fine work. Blue glasses should be worn to protect the eyes, and the gas burners should be furnished with blue shades. He should also avoid stooping and straining, which are apt to induce extravasation of blood. For medicinal treatment, iodide of potassium and purgatives

are recommended. If the patient be strong, and free from anæmia, small doses of mercury may be beneficial. When this affection has become chronic, blisters may be applied to the temples, and chalybeate tonics are of special value.

In cirrhosis, as in other forms of renal disease, the strength must be supported by nourishing diet, and by iron. All care must be taken to avoid exposure to cold, and residence in a warm climate is eminently beneficial.

CHAPTER XIX.

THE COMBINED WAXY AND INFLAMMATORY DISEASE.

MANY cases of waxy disease are complicated with the inflammatory affection of the tubules, and much of the confusion which exists in the minds of medical men with regard to this disease is the result of observers having failed to distinguish between cases of this kind and purely waxy cases. The history of such cases is in general a commingling of the characteristic features of the two. The following instance may suffice as an example of the class.

CASE XXXI.—*Waxy degeneration.—Acute nephritis supervening. Uraemia.—Recovery.—Recurrence of inflammation.—Death.*—Jas. Norval, a baker, aged 34, was admitted to the Royal Infirmary, under my care, on November 1st, 1866. The patient had generally been healthy, but led an exposed life, had at one time had chancre and a bubo, was rather intemperate. He stated that he never suffered from renal symptoms until a few days before he came under my care. Four days before admission, he observed that his ankles were swollen, and as the dropsy increased, he came to the Infirmary. On admission, his legs and feet were œdematous, there was considerable ascites, and some degree of general dropsy. He complained of a shooting pain in the left lumbar region, and had frequent calls to micturition, the quantity passed at a time being small. It was of specific gravity 1013, its reaction acid, its colour smoky, and it contained one-third of albumen. It deposited a sediment, consisting of blood corpuscles, with granular, epithelial, and hyaline tube-casts. There was some œdema of the lungs. The pulse was 47 in the minute, the heart sounds natu-

ral. He was ordered diuretics, consisting of digitalis and bitartrate of potash. On the 3d his urine was 50 oz., but the dropsy was increased. He was ordered to be dry cupped over the loins. On the 5th, his urine had increased to 100 oz., but the amount of albumen was still large, the dropsy, however, was diminished. On the 10th, he injudiciously exposed himself to cold, and on the 11th, his urine was suppressed. The dropsy then increased, and he complained during the forenoon of severe frontal headache, and in the course of the day became comatose. Between seven and eight in the evening, he had a severe convulsion fit, and five fits occurred between that time and ten o'clock. He was then cupped over the kidneys to 10 oz. by my clinical assistant Dr J. W. Paton. Under this treatment he became conscious, and the flow of urine recommenced. At three o'clock, on the morning of the 12th, he had another fit, and complained much of headache. He was ordered a drachm and a half of compound jalap powder. On the 13th, he was somewhat better, the urine 52 oz., of specific gravity 1012, containing one half of albumen. The diuretics were resumed, and on the 17th, he passed 100 oz. of urine. On the 24th, the urine was 90 oz., and dropsy being still considerable, he was ordered to have inhalations of the oil of juniper. On the 25th, the urine was 110 oz. From this time the quantity of urine was considerable, but the dropsy continued. In the course of December, fatty tube-casts prevailed, the bloody casts having totally disappeared, and the epithelial and granular become rare. Early in January, punctures were made with needles in the legs and in the skin of the abdomen, and large quantities of fluid drained away. Under this treatment, followed by tonics, he improved, and became so well that he was able to abscond from the Infirmary in March. In December, he again presented himself, was entirely free from dropsy, was passing, according to his own account, about the natural amount of water, but it contained some albumen.

March 26th, 1868.—Norval again presented himself at the Infirmary. He stated that he had had very good health from the time he left the Hospital until January of the present year. During November, December, and January he was employed in the General Post Office, and walked about twenty-five miles daily. In January he met with an accident that confined him to the

house for a few days, and he lost his employment. Since that time he has been tramping about in search of work, and much exposed to vicissitudes of weather. About the 12th March, he noticed that his feet were swollen, and soon after his hands also became affected. The day before his admission he got wet through, and continued to wear his wet clothes till they dried. In the morning his eyelids were swollen, and the dropsy much increased.

On the day of admission he passed 28 oz. of urine of a smoky colour, highly albuminous. On microscopic examination blood corpuscles, fatty epithelium, and hyaline, granular, and fatty casts were found. There was general dropsy. The tongue was furred and moist; the appetite was good. He complained much of thirst; the abdomen was distended with fluid. The voice was husky (as it had been for eleven years, probably from syphilitic disease.) There were distinct signs of œdema of the lungs. He had a good deal of cough, and expectorated a watery mucus tinged with blood. There was a systolic murmur, loudest at the apex. The pulse was 60, firm and regular. He was treated by means of diuretics and other remedies, but the quantity of urine did not increase, while the dropsy steadily gained. He had no convulsions nor coma, but was affected with restless delirium for some days before his death, and died on April 4th, after expectorating a considerable amount of blood.

Autopsy.—Only the kidneys were examined. The body was dropsical. The kidneys were somewhat below the natural size, and much congested. The capsules were somewhat adherent, and the surface of the organs was granular, the cortical substance atrophied. The tubules of the cortical substance contained a large quantity of fatty matter, and many were filled with recent exudation. The vessels and small arteries were waxy.

I have selected this case as a typical example of the combined waxy and inflammatory disease. The patient was of a syphilitic constitution, and, though he was not aware of it, probably had symptoms of waxy disease long before the occurrence of the inflammatory attack, for which he first came under my care.

That attack was very severe, and he would have died of uræmia but for the timely interference of my assistant. Even after the quantity of urine had risen to 100 oz. daily, the dropsy continued, until it was mechanically removed by puncturing the skin. The quantity of urine remained high to the end of his first residence in the Infirmary, and probably it was excessive during the interval of comparative health which succeeded. On his return, he was suffering from a new and severe attack of inflammation, and from the commencement I regarded the case as almost hopeless. The remedies which formerly saved his life were now powerless. His dropsy increased, and ultimately proved fatal. It is obvious that the inflammatory affection masked the waxy degeneration, and but for the syphilitic history and the polyuria, it might not have occurred to us that waxy degeneration existed, as it was, a degree of uncertainty prevailed, because of his statement that his urine had never been excessive before he came under my care.

The treatment adopted was a combination of that which has been recommended for each of the two diseases from which he suffered. The value of blood letting, purgatives, diuretics, tonics, and acupuncture were well shown in the earlier part of the history.

CHAPTER XX.

COMBINED CIRRHOTIC AND INFLAMMATORY DISEASE.

PARTICULARLY in the later stages of the cirrhotic disease, inflammation of the tubules is apt to supervene. In not a few cases, indeed, renal disease is not detected until the sudden occurrence of the prominent symptoms of nephritis draws attention to the condition. The following narrative may suffice as an example. It is the further history of a man whose earlier symptoms are described as an instance of the cirrhotic kidney. In the former account I showed that under treatment he improved, and was able to leave the Infirmary.

CASE XXXII.—*John Stewart's history continued.—Cirrhotic kidney, supervention of inflammation, death.*—The patient was readmitted to Ward VI., under my care, on the 25th May 1868, five days after his dismissal. After leaving the Hospital he seems to have gone straight off and entered on a debauch. He reappeared in a wretched state, without his boots, and with severe rheumatic pains in his ankles, knees, and elbows. He was ordered colchicum and alkaline diuretics.

May 26th.—Very ill. Complains of intense thirst. Ordered to take a table-spoonful of lemon-juice in water now and then. Urine acid, very albuminous and smoky—sp. gr. 1009. Quantity not ascertained, but on the days immediately following it was found to be—

May 27th, ...	84 oz.
„ *28th*, ...	94 „

May 29*th*, ... 104 oz.
„ 30*th*, ... 68 „
„ 31*st*, ... 60 „

June 1*st*.—Last night the patient began to suffer from considerable difficulty of breathing. Urine diminished in quantity to 54 oz. It is of a smoky hue, and contains blood corpuscles. On heating it, and adding nitric acid, a deposit of albumen forms to the extent of one-half. There is considerable œdema of the walls of the chest and of the loins, and to a slight extent of the lower extremities. He complains of no pain over the region of the kidneys. He does not sleep at night, and is very restless. There is no headache, and the pulse is of moderate strength. To-day he was ordered 2 drachms of compound jalap powder, and a diuretic mixture.

Vespere.—About an hour after the administration of the purgative the patient had four or five copious liquid evacuations, which considerably relieved the oppression under which he laboured. There is still considerable difficulty of breathing, and an anxious expression. There is no dulness on percussion anteriorly or posteriorly, but over all the chest loud sibilant and sonorous rales are heard. Was ordered to have turpentine stupes applied over the chest.

June 2*d*.—Did not sleep well at night, although the breathing was considerably relieved by the turpentine stupes. The bowels have been frequently moved since last night, but little has been passed. The patient complained of hemorrhoids. The urine is diminishing in quantity, and its hue is darker. It contains blood corpuscles. Quantity 53 oz.

June 3*d*.—Patient says he does not think he is any worse. His breathing is still oppressed. The turpentine stupes were repeated last night. Bowels have been often moved, but little passed. The urine is considerably diminished in quantity, and is of a redder colour. Contains numerous blood corpuscles and bloody casts.

Vespere.—Had turpentine stupes applied over his loins in the afternoon. Was dry-cupped over the loins, and ordered to have a hot-air bath.

June 4*th*.—Patient slept a little last night. The hot-air bath caused copious perspiration. The urine is steadily diminishing

in quantity, and looks much more bloody than before. Quantity 24 oz. To-day he was ordered another dose of compound jalap powder.

Vespere.—The patient's bowels have been moved once or twice. Ordered to have the hot-air bath, and to be dry cupped.

June 5th.—Urine not measured, as patient did not pass any except when at stool. The breathing is much the same as before. Ordered to have turpentine stupes applied. He was also ordered to inhale 30 drops of oil of juniper occasionally, and to have the hot-air bath repeated to-night, as it did not act last night, though kept up for a considerable time.

June 6th.—Sibilant and sonorous sounds, with mucous rales, accompany the respiration all over the chest. There is no dulness anteriorly, but posteriorly there is dulness over the base of the lungs. He complains of a pain in the abdomen on each side, corresponding to the course of the ureters. He slept a little last night. The hot-air bath caused copious diaphoresis. He makes water only when at stool, and in very small quantity. He says he does not feel worse, but rather easier. He does not complain of headache, but seems always inclined to dose. There does not seem to be any obtuseness of the mental faculties, but his face has a very anxious expression.

June 7th.—Patient had the hot-air bath this morning, and perspired a good deal. His urine is a little increased in quantity, but still cannot be measured, as it is passed chiefly at stool. There is no headache, but the same anxious expression, and great difficulty of breathing.

June 8th.—Patient to-day looks worse than yesterday. His mental faculties are getting obtuse. He says he feels rather "queer," but cannot say what it is in particular. He complains of headache. He passed a very restless night, and appeared to be rather delirious. His urine is almost suppressed. Before the visit, he had the hot-air bath, but it seemed to excite him, according to the nurse's statement, and it was accordingly discontinued. He was ordered turpentine stupes over the chest, also a purgative of compound jalap powder, and eight ounces of gin.

Vespere.—Patient is comatose. His motions are passed in bed. He cannot be roused, and his pupils are insensible to light. His respirations are stertorous and gurgling—36 in the minute.

His pulse is full—78 in the minute. His skin is moist and warm. The present change came on about 5 P.M. after an attack of vomiting. The patient died comatose at ten minutes to ten o'clock.

Autopsy.—The body was dropsical. The abdomen alone was examined, and more particularly the kidneys. The peritoneal cavity contained a considerable quantity of serous fluid. The liver appeared to the naked eye slightly cirrhotic. Under the microscope many of its cells were found to be fatty, and the fibrous stroma was somewhat increased. The right kidney weighed 4, the left 5 oz. In both the cortical portion was diminished relatively to the cones. The cortical substance was congested, and contained many small cysts. The capsule was slightly adherent. The surface was granular and greyish in colour. The spleen was pulpy, congested, and friable. The bladder contained about 2 oz. of urine. On microscopic examination of the kidneys, the connective tissue was found much increased, and the substance atrophied towards the surface. The epithelium was cloudy and granular, and many of the remaining tubules were blocked up by exudation in some parts recent, in others of older standing, and fatty. There were also numerous points of extravasation throughout the cortical substance.

I have selected this case as a typical example of the combined cirrhotic and inflammatory diseases. Up to the time of his leaving the Hospital the patient had exhibited no symptom of inflammation of the tubules; but when, in the course of his debauch, he was exposed to the weather, such inflammation was lighted up. It appeared that the pre-existing disease rendered fruitless our efforts to relieve the tubules, and the patient gradually became worse, and died a fortnight after his return. Had we not known that cirrhosis existed in his kidneys we could not have diagnosed anything, excepting the inflammation, after it had been established. The only consideration which might have

guided us to such an opinion was the fact that the symptoms were more severe than the amount of inflammation seemed to warrant. The case shows how serious a complication inflammation proves when superadded to cirrhotic disease of the kidney.

In the preceding pages I have sought to show in outline the characteristic features and the appropriate treatment of the three diseases which bear the name of their discoverer. Many points have been passed over, being regarded as matters not essential to such an account as I aimed at giving. Among these I may refer to such exceptional conditions as the occasional occurrence of the different forms without albuminuria, and inflammation affecting only one kidney, and thus inducing a series of anomalous symptoms.* I have endeavoured to indicate what contributions to our knowledge we owe to different authors, but in a subject so rich in literature it is difficult to do so fully.

* See Harley on Albuminuria, p. 20.

SUPPLEMENTARY CHAPTERS.

I.

On the Simple Fatty Degeneration of the Kidney.

ALONG with fatty degeneration of the liver and of the muscular substance of the heart, with or without general obesity, we occasionally find a fatty degeneration of the kidney without any trace of inflammation. In clinical history and pathological characters, these cases differ from those of the second stage of the inflammatory form of Bright's disease, with which they are too commonly confounded. So far as I am aware, we are indebted to Mr Simon* for the first recognition of this affection. He says, "In the domestic cat,—at least in our metropolitan cats,—the tubules of the kidney almost invariably (though I presume abnormally) contain a very large quantity of oil; and I think it probable that the quantity may be artificially increased by interference with the locomotion and respiration of the animal. This is a condition of simple fatty accumulation, analogous probably to the fatty liver of the human subject. Though immeasurably greater in degree than any similar accumulation ever observed in the human kidney, it is attended by no destruction of the

* Lectures on General Pathology, p. 185.
See also Dr Johnson on the Pathology of Bright's Disease, Medico-Chirurgical Transactions, Vol. xxix., p. 15; and Diseases of the Kidney, p 392.

tubules; nor does it often, if ever, interfere with the functions of the organ or with the health of the animal." He states further, that when he first observed this condition he thought it analogous to the scrofulous form of Bright's disease.

Organs affected in this way are generally of about the normal size. Their surface is smooth, and the capsule strips off easily. There is no congestion, and scarcely any stellate veins are visible. The organ is more soft and flexible than natural, and the surface is mottled with numerous deposits of sebaceous-looking material. On section, the relative size of the cortical substance and the cones are preserved, and, beyond a general pallor, there is no change, excepting the abundant deposition of sebaceous-looking material, mostly in the tubules of the cortical substance, but also in those of the cones.

On examining it with a low power (*Plate vii., fig.* 1), the characteristic fatty opacity is well marked, and by careful scrutiny it may generally be made out that the deposit is just within the lining membrane of the tubes, in fact, not in the free cavity of the tube, but within the epithelial cells. The malpighian bodies, the vessels, and stroma are natural. On examining with a higher power (300 to 400 diameters), the points above referred to are much more distinctly recognizable; and when a tubule is cut across, a clear space may be seen within, surrounded by fatty epithelium.—(*Plate vii., fig.* 2.)

This peculiar condition I have had opportunity of studying in many cases, some very well marked, others less so; indeed, in the ordinary work of a pathological theatre we meet with every variety, from the slightest to the most intense. Under what circumstances does it appear? I have met with it only in cases of exhausting disease, such as cancer, and in individuals who appeared to have assimilated an undue amount of oily matter, as was testified by the copious adipose tissue, and the fatty state of the liver and the heart.

I. *Fatty degeneration in connection with exhausting diseases.*

Foerster* remarks that it occurs in cases of long-standing maladies, such as tubercle, cancer, and caries. Dr Otto Beckmann† confirms Foerster's statement, and records the following case:—"In a girl who died at the age of eighteen, in a house of correction, there was found extreme cheesy hepatization of both lungs, with softening and cavities, cheesy infiltration of the bronchial and mesenteric glands, on the right side a recent, on the left an old pleurisy, very extensive tuberculous ulceration of the intestine, pigmented spleen, and very fatty liver. The kidneys were very anæmic, pale yellow, moderately large, with slight injection of the superficial stellate veins. In many of the tubules of the cortical substance and of the cones there was merely debris composed of larger and smaller fatty granules, in others the cells were retained, filled with fatty matter. The malpighian bodies were natural." Beckmann had no information as to the state of the urine.

In illustration of this condition, I subjoin the following case:—

Christina Thorburn, æt. 52, Ward XV., Dr Sanders.—The patient had for some time suffered from cancer of the œsophagus. Never had dropsy, albuminuria, or any symptom of Bright's disease.

Autopsy.—The body was emaciated. There was no dropsy. The muscular substance of the heart was fatty. The right lung was natural; the left was adherent to the pericardium and the thoracic wall. On separating the left lung posteriorly below the root, there was found a gangrenous condition of parts connected with the œsophagus. The pharynx was natural. The œsophagus was affected with cancer for three and a-half inches of its extent, commencing about the root of the lung, and extending to within two inches of the stomach. The cancer was epithelial, extensively ulcerated, and on the left side communicated with a small gan-

* Foerster's Pathologische Anatomie, Bd. ii., s. 520. † Virchow's Archiv, Band. xi., s. 65.

grenous cavity, bounded on front and at the side by the lung, posteriorly by the thoracic wall. The stomach was much contracted. The intestine was natural. The liver was extremely fatty. The spleen was natural. The kidneys were of normal size; their surface smooth, but pale, mottled with sebaceous looking material. The capsule stripped off readily. On section, the relative size of cortical and conical substances was natural. Many of the convoluted tubules were filled with sebaceous looking material. The vessels and malpighian bodies were natural. The epithelium in the affected tubules was loaded with fat, but the lumen of many of them was ascertained to be clear, surrounded by the fattily degenerated cells still in situ. There was no trace of exudation in the tubules.

In this case the amount of fat was such that it was apparent even to the naked eye, and in microscopic examination was as abundant as I have ever seen in the second stage of the inflammatory affection.

II. *Fatty degeneration in connection with Senile Marasmus.*

Dr Rosenstein believes that he has seen this result from senile marasmus, and gives the following case in illustration:—

A woman, aged 73, came into the Hospital on account of an injury to the face, and prolapsus ani. She stated that she had always been healthy, and exhibited no symptoms of any malady beyond that for which she sought admission. The prolapsus was great, and the exposed mucous membrane always discharged fluid and mucus. The urine was natural in quantity, and contained no albumen. In the last three weeks of her life she had diarrhœa, slight œdema of the legs, and albuminuria, but no casts. At the autopsy the heart was found somewhat atrophied. The spleen was small, its capsule wrinkled and thickened. The liver was considerably diminished, with sharp fibrous edges; the surface smooth, pale yellow in colour, and fatty. The kidneys were small; the capsule adherent; the surface finely

granular, but without trace of congestion. On section, the cortical and conical parts were difficult to distinguish, of a pale yellow colour throughout. The epithelium of the tubules was everywhere fatty; the malpighian bodies were natural.

In commenting on the case, Rosenstein remarks, that keeping in view the history, the symptoms during life, and the concomitant wasting of the liver and spleen, he cannot suppose an inflammatory condition to have existed, but, on the contrary, must conclude that it was due to senile marasmus. He refers the œdema which came on before death to the exhausting diarrhœa. If this view be correct, we have here another cause of the fatty degeneration under consideration.

III. *Fatty degeneration from excess of fatty food.*

Certain observations make it appear that this condition may also arise from the introduction of an excess of fatty matter into the system. Dr Beale has found it in the kidneys of cats who lived in breweries, and thus enjoyed peculiar abundance of hydro-carbonaceous food, and little exercise. In the dog, the ox, and the calf, the kidney normally contains much oily matter. Dr Lang * of Dorpat, made the interesting discovery, that even in men, especially with very fatty food, some fat passes into the urine, as is almost constantly observed in cats and dogs. On examination of the kidneys, Lang found the fat granules in the cells most abundant in the convoluted tubules, a fact which Kölliker† also observed in the kidneys of sucking animals. Beckmann found it most marked in some cases in the straight tubules. Reinhardt describes two cases in which the epithelium was very fatty, and in which there was no symptom of Bright's disease during life. The best marked case

* A. Lang, De Adipe in urina et renibus, etc. Dorpat 1852. Quoted by Beckmann and by Rosenstein, loc. cit.

† Würzburger Verhandlungen, vi., s. 183. Quoted by Beckmann.

of this kind with which I have met, was in a lady of somewhat intemperate habits, who had become very obese. She had never suffered from any renal symptoms. Her body was very rich in adipose tissue, the abdominal wall, the mesentery, the subcutaneous tissue generally, and the heart being all loaded with it. The muscular substances of the heart was fatty, and the liver and kidneys presented excellent examples of extreme fatty degeneration. The epithelium in the kidneys, for the most part, adhered to the walls, and the tubules, when cut across, showed their lumen clear, and surrounded by the degenerated epithelium.

It appeared in this case that the excess of fatty or carbonaceous matter taken into the system had led to the deposition in the epithelium.

We are thus entitled to conclude that we may have the simple fatty degeneration of the kidney in connection with exhausting disease, old age, or with excess of fatty food. In the two former cases it is probably a true degeneration, in the latter, an infiltration or deposition.

II.

*On Acute Atrophy of the Kidney, a condition sometimes co-existing with Acute Atrophy of the Liver.**

THE frequent co-existence of fatty degeneration of the kidneys with acute atrophy of the liver is generally recognised; and most pathologists are prepared to admit that the process in the kidney is identical with that in the liver. It appears to me further that the kidneys may be primarily and mainly affected, the liver secondarily and to a less degree.

The characters of the kidney in both sets of cases are as follows:—They may be of the natural size, rarely somewhat enlarged, and in most cases smaller than normal. I have found the organs to weigh together between 6 and 7 oz. They are flabby and congested, and sometimes blood is extravasated in the cortical substance. The cortical substance, and, to a less extent, the cones, present a dense consolidated, sometimes a sebaceous-looking appearance.

On microscopic examination, the tubules in the cortical substance, and frequently also in the cones, appear opaque, as if distended with fine injection, and when a higher power is used, a series of changes may be traced identical with those which we find in the cells of the liver in cases of acute atrophy. Some are opaque, swollen, and cloudy, their nuclei obscured by a brownish material, which is, however, albuminous in character, clearing up under acetic acid. Others are crowded with fatty globules of various sizes, others are so broken down that they are represented only by molecular

* The substance of this chapter appeared in two papers read before the Medico-Chirurgical Society, and recorded in the Edinburgh Medical Journal for October 1865 and January 1866.

debris. In transverse sections of the tubules these changes may be particularly well seen. The malpighian bodies present no abnormalities, excepting that they are sometimes congested, and sometimes the seat of extravasation.

In illustration of the disease as it affects the liver and kidneys together, I select the following case :—

Mrs H., æt. 35, was admitted to the Royal Infirmary, almost moribund, on 11th February 1865. She was sent to one of Dr Warburton Begbie's wards, but died very soon after admission. Her history was imperfectly ascertained, as she was comatose at the time of her admission ; but from enquiry among her friends who had been with her before she went to the Hospital, I ascertained the following facts :—She was in the sixth month of her fifth pregnancy, and was leading an unhappy life in consequence of domestic quarrels ; but it was not known that any special disagreement had preceded her illness. She was in good health until a fortnight before her death, but then complained to her neighbours of a strange feeling of uneasiness which she felt and feared, but could not define. She gradually became worse, and began to vomit yellow matter like the yolk of an egg. She then also became affected with jaundice, and this gradually deepened. Her bowels were constipated. She never was drowsy or delirious, nor did she vomit blood nor pass it at stool until the morning of the 11th February, when she became much worse, vomited a large quantity of blood, became very drowsy, and could not be roused. In the afternoon, Mr Furley was called to see her, and, by his direction, she was removed to the Infirmary. The movement roused her, and she became for a little time conscious. On admission to the Infirmary she was shivering and complained of cold. She vomited blood almost incessantly. She passed her fæces in bed ; they were dark-coloured and fluid. She made water, but it could not be collected for examination. About eight o'clock, she became delirious, violent, and very noisy. This continued until midnight, after which she was comatose. Labour commenced about eight o'clock ; the waters came away about nine. She was delivered of twins just as she was dying, at seven o'clock on the morning of the 12th.

Autopsy.—The body was examined thirty-one hours after death. It was moderately well nourished. The skin was icteric and somewhat dusky. There was considerable hypostatic congestion, but no subcutaneous extravasation of blood. The muscles were dry; the blood dark and fluid. All the internal organs were jaundiced. There were patches of extravasation under the visceral layer of the pericardium, particularly over the left auricle, and towards the upper part of the left ventricle. The heart was moderately contracted, contained no clot, and very little blood. The muscular substance was somewhat pale; the valves were natural. The aorta was natural. The lungs were congested and somewhat œdematous, particularly at their lower and posterior parts. The liver was reduced to one half its natural size; but, except being somewhat flattened, it retained its ordinary form. It weighed 1 lb. 7½ oz. Its surface was not shrivelled. Beneath its capsule and throughout its substance there were numerous ecchymoses, and small patches of an ochre yellow colour, but the mass of the organ was of a dark reddish brown hue. The outlines of the lobules were not recognisable. The gall-bladder was contracted, and contained a little grey inspissated mucus.

On microscopic examination of a scraping from a cut surface of the liver, a large quantity of debris and fatty matter, with cells in different stages of alteration, some full of oil globules, and containing a few bright ochre-yellow granules, and others full of dense granular matter, not fatty, were found. All the cells were, without exception, considerably enlarged, and denser than natural. No natural cell was observed. The amount of bile pigment found both in the cells and debris was less than is usual in such cases.

On examining sections, it was found that the cells in the outer part of the lobules were almost completely destroyed, while those towards the centre were larger, denser, aud more opaque than natural, and the amount of oil was greater towards the margins, where the destruction of cells was taking place. The system of vessels and the fibrous stroma of the organ were not destroyed. It was easy to make sections, and the sections were easily washed without their giving way. In the sections a considerable amount of bile pigment was seen scattered about.

Some demonstrations were made of the blood in the liver, but

neither in it nor in the substance were any crystals or balls of tyrosin or leucine observed.

The spleen was enlarged, weighed 8 oz., was soft and pulpy. There were some points of extravasation in its substance and beneath its capsule.

The suprarenal bodies were somewhat enlarged and partially disorganized, but their microscopic structure was not ascertained.

The kidneys were somewhat enlarged, weighed together 10½ oz. Their cortical substance was dense and pale. The capsules was easily stripped off. On microscopic examination, the tubules, both straight and convoluted, were seen to be full of exudation, and presented the appearance of having been very successfully injected with some dark matter. The malpighian bodies and vessels stood out clear and transparent among the tubules. With a higher power the vascular structures appeared natural; the dark matter occupying the tubules was found to be composed of exudation into and between the cells. Very few tubules remained healthy; in some the epithelium was swelled, thickened, in a state of cloudy swelling, and here and there it was loaded with fat granules. In some parts the outlines of the renal cells could not be made out; the tubules were full of a dense homogeneous granular matter, containing numerous oil globules.

The æsophagus was natural. The stomach was distended, and contained a good deal of dark uncoagulated blood. Its inner surface was coated with slimy mucus; its walls were thickened, and in the mucous coat there were numerous catarrhal ulcers, mostly along the greater curvature, and on the anterior and posterior walls. Extravasation of blood existed about the bases of some of the ulcers.

The intestine contained almost no bile, but some altered blood, and a good deal of hard nearly clay-coloured fæces. The peyerian patches and solitary glands were swelled and prominent, particularly those about three feet from the lower end of the ileum. The large intestine was natural.

The pancreas was natural.

The mesenteric glands were somewhat enlarged.

The uterus was large, about seven inches in length, moderately

contracted; the cervix was long, and a plug of bloody mucus projected from the os.

Both ovaries were much scarred, and each contained a distinct corpus luteum.

The fallopian tubes, particularly their fimbriated extremities, were congested.

The skull was natural. There was a little subarchnoid effusion. The brain was firm, somewhat congested throughout; it weighed 2 lb. 6½ oz. The ventricles were not dilated. There were a few small cysts in the choroid plexus.

The bladder contained nearly 16 oz. of dark amber-coloured urine, which was carefully removed and sent to Dr Arthur Gamgee, who kindly analyzed it, with the following result:—

"The urine was of an intensely jaundiced hue, and exhibited, in a very characteristic manner, the reaction of bile pigment. Its reaction was acid. It was divided into two portions—A and B.

"The portion A, measuring 200 c. c., was precipitated with neutral and basic acetate of lead, and the fluid filtered. The lead precipitate was reserved for the analysis of the bile acids.

"The filtrate was treated with a stream of sulphuretted hydrogen gas, in order to precipitate the excess of lead, and the clear and almost colourless filtrate thus occasioned was concentrated at a very gentle water-bath heat, and then placed aside in a cool place. At the end of twenty-four hours an abundant crystallization had taken place. The crystals, when examined under the microscope, were found to consist of the most characteristic needles and tufts of tyrosin tinged of a light yellow colour; they were separated by filtration, and dissolved in boiling water. On cooling, the water deposited a nearly snow-white mass, composed of beautiful needles of tyrosin; these were again crystallized from a solution in boiling water, and then dried. In the process of drying they contracted very much. When strongly heated they burned away, without leaving a trace of ash. They exhibited, in a most characteristic manner, the chemical reactions of tyrosin.

"The fluid from which the tyrosin had been separated was evaporated to a syrupy consistence, and set aside for some days. When examined, it was found to contain, in addition to much tyrosin which had separated, characteristic masses of leucine.

"The precipitate which had been obtained by precipitating the urine with acetate of lead was suspended in water, and a stream of sulphuretted hydrogen passed through it. The fluid was filtered and evaporated at a gentle heat. The residue was dissolved in water, and tested by Pettenkoffer's test for bile acids, but none were found.

"The smaller portion of urine, B, was employed to determine the presence and quantity of the more usual urinary constituents, and the results of the analysis are tabulated below.

"The points which specially call for notice are the following:—1*st*, The urea was determined by Liebig's method. As tyrosin is, equally with urea, precipitated by nitrate of mercury, the results of the analysis are obviously not perfectly correct, the urea and tyrosin having in fact been estimated together. As the tyrosin appears to have been present in very large quantity, it may be assumed that the amount of urea was actually very small. 2*d*, The amount of uric acid could not, from the excessively small quantity of urine which was obtained, be estimated; only traces of it, however, existed. 3*d*, The urine contained absolutely no chlorides, only the faintest trace of sulphates, and the earthy phosphates were absent. The only salts present were thus alkaline phosphates. This fact, which was brought out by the qualitative analysis, was also borne out by the quantitative; for the amount of phosphoric acid which was found, when calculated as phosphate of potash, is almost identical with the amount of ash as found by direct ignition."

Table showing the Result of Analysis of Urine.

Specific gravity of urine at 60° Fahr.,	1018·82	
Reaction acid.		
Total solids in 100 c. c.,	2·194	
Urea and tyrosin in 100 c. c.,	1·80	gramme.
Leucine, uric acid, and colouring matter in 100 c. c.,	·1415	...
Albumen,	·0116	...
Salts,	·2525	...
Phosphoric acid,	·204	...

This case affords a very good example of the acute atrophy of the liver, but it seems also entitled to special notice in

regard to the state of the urine, as recognised by Dr Gamgee, and the peculiar condition of the kidneys.

I. *The State of the Urine.*—The points which appear to be most important are,—1*st*, That notwithstanding the presence of a large amount of abnormal ingredients, the *total solids* were considerably below the natural quantity, and thus the functional activity of the kidneys was shown to be diminished; 2*d*, That *leucine and tyrosin* were the chief abnormal ingredients, as is usual in this disease; 3*d*, That a little *albumen* was present; 4*th*, That notwithstanding the extreme jaundice, *no bile acids* were found,—a fact which, at first sight, seems to confirm Dr Harley's view that these constituents are awanting in cases of jaundice from suppression; but this point cannot be deemed important, when we consider that only Pettenkoffer's test was employed, a test which chemists do not now consider satisfactory; 5*th*, that the *urea* was probably much diminished in quantity, and only traces of *uric acid* were found, while in most febrile and hepatic affections these elements are found in excess; 6*th*, That the *chlorides and earthy phosphates* were entirely absent, and that only the faintest trace of *sulphates* was found.

It is evident that some of these altered conditions of the urine depended upon the state of the kidneys and some upon that of the liver and the system generally. There can be little doubt that the leucine and tyrosin resulted from changes in the liver, and that they were eliminated from the kidneys along with the water, at a time when the renal epithelium had become incapable of separating from the blood the natural solids of the urine. The absence or diminution of some of the elements—as the chlorides, earthy phosphates, and sulphates—may have depended upon the state of the system generally, but almost certainly the diminution of the urea depended upon the peculiar condition of the kidneys, for Frerichs has found that in this disease its quantity in the system is rather increased than diminished.

It is to be regretted that the only urine obtained for analysis was that found in the bladder after death. It is very probable that, had we been able to examine the urine for some days before death, we might have found, as did Frerichs in some of his cases, a progressive diminution of the urinary solids, coincident with changes in the kidneys. It seemed impossible that kidneys in the state in which this woman's were found could be capable of any secretion at all; and, from this consideration, I think that the urine which was in the bladder had been secreted some time before, but retained in consequence of the torpid state of the nervous system. I have seen at least one case in which suppression of urine had occurred, in the course of pyæmia, and in which the kidneys were much less affected than they were in this instance.

But though we cannot in the meantime speak positively upon these points, it is evident that in every case of this disease the urine should be carefully examined, and that in treatment constant attention should be paid to the state of the kidneys.

II. *The Affection of the Kidneys.*—The state in which we found the kidneys specially deserves attention. A very copious exudation occupied the uriniferous tubules, and the epithelium was affected in the same way as the cells of the liver. Some were swelled, dense, opaque, granular, some extremely fatty, and some had broken down and disappeared, —a series of changes exactly corresponding with those of the liver. But for the firmer support which the stroma of the kidney and the walls of the tubules affords, we might expect a rapid wasting of the kidney like that which occurs in the liver.

The complete identity of the appearances met with in the two organs satisfies me that they were really affected in the same manner, that the morbid processes were identical, and that we cannot regard the renal affection as a consequence of the hepatic.

In illustration of the disease affecting primarily and chiefly the kidneys, I select the following case:—

M. M., aged 22, was of dissipated and abandoned habits, but always enjoyed good health till the commencement of this illness. In October 1865 she was far advanced in pregnancy, and on the 20th of that month, without known cause, she became suddenly ill, with symptoms which were referred to inflammation of the uterus. But as no satisfactory treatment was possible where she resided, and as she was becoming rapidly worse, she was sent to the Maternity Hospital. She was admitted on Friday evening, 22d October. She was then very weak, and was vomiting stringy mucus, with streaks and clots of blood, and now and then matter like coffee-grounds. This continued, notwithstanding the employment of suitable remedies, until Sunday morning, October 24, when she began to sink rapidly. At that time premature labour was brought on by direction of Dr Graham Weir. She was delivered of a putrid child about 4 P.M., and died at 8 o'clock the same evening. She was never unconscious, remaining quite intelligent until within a few minutes of her death. She had also no convulsions, nor any distinct delirium. She passed urine several times after her admission, but it was in small quantity, and its qualities were not ascertained.

Autopsy forty hours after death.—The body was well nourished. There was no dropsy. Decomposition was rapidly advancing. The pericardium was natural. The heart contained a moderately firm clot, but the blood generally was dark and fluid. The muscular substance of the heart was flabby, but the valves were healthy. Both lungs were congested throughout, particularly towards the base posteriorly. There were traces of old tubercle at the left apex. The liver was of natural size, soft, and flabby, but not friable; the outlines of its lobules were distinct. The spleen was pulpy. The kidneys were small, and weighed together $6\frac{3}{4}$ ounces. They were flabby, congested, and in the cortical substance there were numerous extravasations. That part was not increased, but rather diminished in size, and contained some yellowish-white deposit, which was rendered the more distinct by the surrounding congestion. The stomach contained some dark green matter, like altered blood; its mucous membrane was catarrhal, and was perforated by numerous minute ulcers. Some parts were deeply congested, and at others there were patches of

extravasation. The upper part of the small intestine contained bile and altered blood. The coats throughout were normal. The large intestine was natural. The ovaries were natural. The uterus presented the ordinary appearance of one recently delivered, and was moderately contracted. The bladder contained scarcely any urine; its coats were normal. The head was not examined.

Microscopic examination of the Kidneys.—The whole organ was found to be altered. The tubules, both in the cortical substance and in the cones, were opaque, and appeared as if distended with fine dark injection. On examination with a higher power, the distention of the tubules was found to depend partly upon enlargement of the epithelial cells and partly upon free exudation, which occupied the cavity of the tube. The cells were in some parts opaque, of a brownish colour, and full of dense granular matter, which obscured the nuclei; in others, they contained fatty granules of large size, surrounded by numerous firm fatty molecules; in others, they were represented by groups of oil globules, enclosed by an indistinct cell wall, which broke down under very slight pressure. So great was the friability of these fatty cells, that when a scraping of the kidney was examined, the pressure of a light covering glass sufficed to break down the tube-casts to a fine molecular debris. In transverse sections of the tubules the changes were well seen; in some the epithelium enlarged, dense, brown, filling up the lumen; in others, the opaque matter replaced by fat, the outlines of the cells remaining entire; in others, the cells broken down, and represented by groups of fat granules; others were entirely empty.

The condition of the stroma was also interesting; it was somewhat enlarged, and contained numerous fatty granules. Many of these were arranged in little groups and lines, and appeared as if situated in the connective tissue-corpuscles. The corpuscles were certainly somewhat enlarged, from which it appeared that the lesion was not confined to the tubules.

From the fact that, although every tubule which was seen was distended, the cortical substance was diminished rather than increased, and the organs were below their average weight, it is evident that considerable wasting of the organ must have taken place, and this we can well understand when we think of the

rapid fatty disintegration which was going on. The distinct diminution of bulk of the organs, and the rapidity with which it was taking place, seem to warrant the application of the name "acute atrophy."

The very striking similarity between the conditions just described and those accompanying acute yellow atrophy of the liver cannot be overlooked, whether we regard the general appearance of the organ or its microscopic characters. The whole series of changes, from the exudative infiltration into the complete disintegration of the cells, was identical with that found in the case of Mrs H., recorded above.

Microscopic examination of the Liver.—Under a low power the hepatic cells were seen to be unusually opaque, while towards the margin of each lobule there was a dark zone of fatty degeneration, and outside of that—*i. e.* at the point of contact of neighbouring lobules—there was a clear space in which no cell was visible, only a little granular debris. Under a higher power, the cells were found to present exactly the characters of those met with in acute yellow atrophy—that is, some were enlarged, opaque, full of dense granular matter; others were fatty; others were in an advanced state of fatty degeneration, extremely friable, easily broken down. These different stages were also distributed in the lobules, just as was described in my former case—the swollen infiltrated cells being in the centre, surrounded by those in a stage of degeneration, these again surrounded by the space in which no cell element remained. There was no breaking down of the stroma of the organ, nor any marked bile-staining of the cells, nor retention of bile in the ducts.

It may be suggested that this change was an ordinary fatty degeneration, occurring in its usual seat, towards the margin of the lobule, and not at all to be wondered at, considering the character and habits of the patient. But I think it was something more—firstly, because the appearance of the fatty cells was unlike what we ordinarily see; secondly, on account of the peculiar opacity of the cells in the centres of the lobules; thirdly, because fatty degeneration never leads to destruction of the cells, as was seen in the periphery of the lobules, while in all these points the appearances are characteristic of the acute yellow atrophy.

It appears to me that we are entitled to regard this case as an example of a disease essentially identical with acute yellow atrophy, for the following reasons:—

1*st*. The pathological appearances in the kidneys and liver were the same as those met with in acute yellow atrophy.

2*d*. The leading symptoms of this case,—the peculiar malaise, the sickness and vomiting of blood, the rapid sinking, the congestion of the lungs,—are among the chief of those met with in the other disease.

3*d*. The circumstances of the patient. She was advanced in pregnancy, was leading an exposed and wretched life,—two conditions very commonly associated with the other affection.

From these considerations, my view seems to be well established; at the same time, three important symptoms were entirely awanting, viz., delirium passing into coma, icterus, and diminution of the volume of the liver.

The absence of nervous symptoms, such as delirium, coma, and convulsions, is very strange, considering the condition of the kidneys; for with that condition one would have expected well-marked uræmic symptoms. The bladder was empty, and the kidneys were obviously incapable of secretion. But the symptoms of neither the acute nor chronic form of uræmia were present. This can, I think, be explained only on the hypothesis, which is consistent with the other facts of the case, that the patient died before there was time for the uræmic symptoms to be developed.

The icterus and the diminution of the volume of the liver often occur late in cases of acute atrophy, sometimes not at all; and it may well be believed that, in this instance, death took place before there was time for their manifestation.

In regard, again, to the symptoms which were present, this case seems to teach an important lesson; for the hæmatemesis, which is so frequent a symptom in acute yellow atrophy, occurred here, although the liver was comparatively little diseased; thus showing that it is not dependent on the hepatic affection. May it not be the result of a blood-poison?

If it be admitted that this case is of the same nature as acute yellow atrophy, it certainly gives the *coup de grace* to the theory which would make the renal affection in that disease secondary to the hepatic. It, at the same time, remarkably confirms the opinion that acute yellow atrophy is a blood disease. The general appearance of the body, the marked hypostatic congestion and rapid decomposition, the softness of the spleen, the darkness of the blood and its imperfect coagulation, the occurrence of identical morbid conditions in the liver and kidneys, and the peculiar nature of the process in both organs, differing, as it does, from ordinary inflammation and from ordinary fatty degeneration, and the peculiar circumstances under which it occurred, all point strongly to some cause operating on the system from the blood.

The case, then, supplies an additional point in the history of acute yellow atrophy, and decidedly supports the view of its being dependent on a blood-poison.

The only similar cases that I have found are three published by Rokitansky in 1859, under the name of fatal steatosis of the liver and kidneys,* and two by Dr Hecker of Munich, in 1867. I shall not detail Rokitansky's cases, but merely mention certain general facts in regard to them. All were females, unmarried, aged 23, 38, and 8 years respectively. Two had been in depressed spirits. All were stout, well-nourished, and died after about a week's illness, during which the leading symptoms were—fever, headache, vomiting of dark matters, convulsions, and coma; in two there was slight icterus. On post-mortem examination, ecchymoses were found in many of the internal organs. The livers were large, pale, flabby, and fatty; the kidneys were also large, fatty, congested, and mottled with extravasated blood; the urinary bladders contained scarcely any urine; in two, the stomach and intestines contained altered blood.

* Ueber lethale Leber-und Nieren Steatose, von Prof. Rokitansky. Zeitschrift der k. k. Gesellschaft der Aerzte zu Wien, Aug. 1859.

Microscopic examination showed in all three cases extreme fatty degeneration of the liver and kidneys.

Rokitansky distinctly recognises in these cases a poisoned condition of the blood, which, however, he regards as a consequence, not a cause, of the hepatic and renal affections; and, in accordance with a favourite view, he regards the renal affection as secondary to the hepatic. He refers the fatty condition of the liver to that state of the system in which an excess of fat is present, and a tendency to fatty deposition exists, and having excluded, with regard to the kidneys, the theory that it might depend upon Bright's disease, he concludes by saying, "There can be little doubt that the steatosis of the kidney was secondary to the primary steatosis of the liver; and just as little doubt from the diminution of urine consequent upon kidney disease on the one hand, and the slight degree of cholæmia on the other hand—that the sudden development of the fatal symptoms depended upon uræmia. It would accordingly appear that fatty degeneration of the liver may occur in an individual inclined to excessive fatty deposit, and to this, sooner or later, a fatty condition of the kidney may be superadded, both of which, gradually and unobserved, may attain such a degree that secretion of bile and urine is suspended, and death supervenes suddenly, after the appearance of slight icterus, by uræmia and an hæmorrhagic decomposition of the blood."

Notwithstanding the profound respect to which every opinion of Rokitansky is entitled, I would venture to suggest that the narratives are capable of another interpretation, especially when examined by the light of the remarkable cases which have just been described. It is very difficult to believe that so general and important an affection of the liver and kidneys should have gradually advanced, and yet for long led to no symptom; still more is it difficult to understand how so insidious an affection should have suddenly produced such violent symptoms, how the uræmia should have been unaccompanied by any trace of dropsy, or should have led to a hæmorrhagic condi-

tion of the blood; and, on the other hand, all the symptoms correspond closely with those of acute yellow atrophy, and many of them with that peculiar variety of the disease which I have described. These cases, indeed, seem to constitute a connecting link between the affection in its ordinary form, in which the liver is mainly involved, and the peculiar variety described above, in which the kidneys are the primary seat of disease.

I am indebted to my friend and colleague Dr Matthews Duncan for drawing my attention to Dr Hecker's * observations on this subject. His attention had been directed to it by observing a typical example of acute atrophy of the liver. And he found that in puerperal women a severe and rapidly fatal disease sometimes arises, which, with obscure symptoms—without jaundice or hemorrhage from the bowel—is first recognised at the dissection as consisting of a fatty degeneration, which certainly must have occurred at the soonest towards the end of pregnancy. Appearances corresponding with these he has sometimes met with in newborn children. He records the following case:—

A pale, thin woman of twenty-one was admitted to the Hospital at half-past two on the afternoon of Dec. 22d, 1866. She came on foot. Eight days before she had been examined, and the internal os found dilated. At 3 P.M. on the 23d, she had a severe rigor, followed by heat, quick pulse, breathlessness, and severe epistaxis. At 5.30 she was delivered of a child, which appeared to have been eight days dead. Considerable bleeding followed the removal of the placenta. The patient said she felt well, but was pulseless; and at 6.30 she suddenly died.

On post-mortem examination, there were some slight extravasations within the cranium. The lungs were congested and œdematous; there was no trace of embolism. The bronchi contained some bloody mucus. The heart was soft, its fibres natural. Extravasations existed here and there

* Monatschrift fur Geburtskunde und Frauenkrankheiten, Band xxix., s. 321, und xxxi., s. 197.

beneath the pericardium. The substance of the liver was dark brownish red, the stroma somewhat increased; the hepatic cells were granular. The spleen was considerably enlarged and pulpy. The mesenteric glands were swollen. Both kidneys were in a state of cloudy swelling; the tubules were filled with granular matter. There were recent extravasations in the pelvis, and in the walls of the bladder. There was no air in the veins; the uterus was flaccid, blood was extravasated into its substance.

Dr Hecker ascribed the death to dissolution of the blood, a result of renal and hepatic disease.

He gives cases of similar acute atrophy in human infants, as well as in the young of some of the lower animals.

Another case which he has more recently recorded is as follows:—On July 16th, 1867, he was informed of the death of a patient who had been delivered on the 15th without accident. She had suddenly become breathless, and died 28 hours after the confinement. At the examination, 23 hours after death, there was little decomposition, and no dropsy nor jaundice, but numerous ecchymoses over the abdomen. In the thorax there was a good deal of yellow serum. The lungs were congested, and somewhat œdematous, with numerous subpleural extravasations. The heart substance was brittle; beneath the endocardium there were many ecchymoses; valves natural; no embolism of pulmonary artery. The liver was very yellow, not reduced in size, soft and fatty. The spleen was enlarged, pretty hard, almost waxy-looking. The kidneys were distinctly in the second stage of parenchymatous inflammation; capsule readily separable; parenchyma swollen; cortical substance yellow. The uterus weighed 1100 grammes; its walls thickened; the mucous membrane easily separated from the muscular substance; in the cervix there had been copious hemorrhage; there was a corpus luteum in the ovary. The walls of the large intestine were infiltrated with extravasated blood which had not made its way through, the fæces being of natural colour.

In the stomach and duodenum a similar condition existed. The lymphatic glands were swollen. On microscopic examination the fatty degeneration of the heart, liver, and kidneys were in parts very distinct, especially in the kidneys.

These different cases appear to me to illustrate a series of diseases, or phases of one disease, resulting from poisons generated in, or introduced into, the system; and some are not very far removed from what would be universally recognised as examples of inflammatory Bright's disease. I believe that their further study may lead to valuable results.

I have long held that all these processes are results of blood-poisoning, and in addition to the clinical and pathological evidence which the cases narrated contain, it appears to me that the recent observations on the state of the viscera in poisoning with phosphorus confirm this view. We can, with that substance, artificially produce a disease scarcely distinguishable from some of the cases to which I have referred. Dr Ranvier,* one of the most recent writers on the subject, states that the kidneys of those poisoned with phosphorus show the characters of complete and diffused steatosis. He describes some of the tubules as being filled with an albuminoid substance; others in a state of extreme fatty degeneration, which is occasionally so marked that the epithelial cells have entirely disappeared. These conditions are identical with those above described. Such results strongly corroborate the opinion that poisons in the blood induce the visceral lesions.

* Journal de l'Anatomie, No. 2, 1867.

III.

On the Nature of the Waxy or Amyloid Degeneration.

In the present state of science it appears not to be out of place to devote a supplementary chapter to an account of the views entertained as to the nature of the waxy or amyloid degeneration. I shall not enter into the history of its discovery and gradual elucidation further than may be necessary for explaining the views entertained by different observers.*

The degeneration consists in the replacement of the natural tissues by a peculiar pale, dimly-translucent material, which assumes a characteristic coloration with iodine and other coloring matters, is little liable to undergo decomposition, and resists the action of most re-agents.

It tends to affect cells, small arteries, capillaries, and non-voluntary muscular fibres; and, in a great majority of cases, wherever it appears in the body it will be found to affect these structures. In the arteries it first attacks the transverse fibres of the middle coat, and afterwards extends to the other elements of the wall. It may be well to state, in a word or two, its mode of manifestation in different organs. In the *Liver* it occurs, 1*st*, as a general affection of the cells, rendering the organ large, pale, waxy-looking, cutting cleanly, and showing at the sharply-angular margins a degree of transparency. On microscopic examination the cells are found considerably enlarged, dimly translucent, with their nuclei obscured. 2*d*, As a general affection of the smaller branches of the hepatic artery, and apparently also of

* I would refer the reader to an article in the Ed. Med. Journal for 1868, in which the early history of its discovery in Edinburgh is clearly set forth, also to an inaugural dissertation "Ueber die Amyloide Degeneration," by Dr Arnold Pagenstecher, Wurzburg, 1858.

the portal vein, the cells being unaffected, and the condition only distinct when microscopically examined, and tinged with iodine. 3*d*, As a general affection of the vessels and cells in certain individual parts here and there forming masses which closely resemble bees-wax. This condition has been rarely met with.* 4*th*, A diffused affection of the cells and vessels throughout the organ. In the *Spleen* it affects the smaller arteries, the malpighian bodies, and more rarely the pulp. In the *Kidneys* it affects the malpighian bodies and small arteries, rarely extending to the basement membrane of the tubules, or to their contents, but constantly leading to secondary infiltration of the organ, as has been described in the body of the work (*Chap.* VIII.) In the *Stomach* it affects specially the small arteries. In the *Intestine* the small arteries, the epithelium covering the mucous membrane, the muscular substance of the mucous coat and of the villi (Brücke's muscles) are its chief seats, but it never affects the substance of the tissue of the villi, and rarely the muscular substance of the middle coat. In the *Pancreas* it affects the small arteries, and not, so far as I have seen, the secreting structure. The arteries are also most frequently affected when the disease spreads to *Lymphatic Glands*. The *Muscular Fibres* of the uterus and the arteries of the vaginal wall are occasionally affected. I have never seen it in the lungs nor in the brain, though some observers speak of it in both these situations. In the *Heart* I have seen distinctly waxy vessels. I am informed that Dr Gairdner has found it in cancer. Dr Bennett† states that he has seen it in the *placenta*. In the *Skin* it sometimes occurs. Indeed, it would appear that, while the above-mentioned are its chosen seats, it may occur anywhere in the body.

The peculiar colorations which the waxy material undergoes have attracted much attention. Virchow‡ was the first to

* British and Foreign Medico-Chirurgical Review, Oct. 1864. Die Krankhaften Geschwülste, Bd. ii., s. 430.

† The Principles and Practice of Medicine, 5th Ed., p. 250.

‡ Virchow's Archiv, Band vi.

show that with iodine it assumes a peculiar mahogany red hue, and that the further addition of sulphuric acid changes the colour to violet. The reaction with iodine is, in my opinion, the best test of this material, being easily produced, and quite unmistakeable. That with sulphuric acid is less reliable, for it often fails to produce a blue, although generally a purple colour appears. Some writers deny the possibility of getting the blue colour described by Virchow. I have seen it perfectly distinct, but have, on the other hand, so often failed to obtain it, that I generally content myself with the iodine test. This may be employed simply by pouring a little of the aqueous solution (liquor iodi of the British Pharmacopœia) over the surface, when the naked eye will at once detect the appearance; or it may be applied to microscopic sections. Dr Bennett has shown that equally distinct coloration may be obtained with other materials, such as carmine and magenta, but none is so convenient for use as the iodine.

Many views have been advanced as to the chemical composition of this material. Heinrich Meckel* conceived that it was cholesterin, and proposed to term the degeneration the cholesterin disease. He thought it in some way related to the fatty degeneration. His view has been totally disproved by the reaction already mentioned—cholesterin behaving with re-agents very differently from the waxy material—and by the chemical analyses to which I shall presently refer. Virchow at one time held that the waxy material was allied to cellulose, and therefore proposed to term it the amyloid degeneration. This view also has been completely disproved. Its origin, however, is still worthy of attention. It had long been thought that no starchy material existed in the animal body, that, in fact, starch and cellulose were characteristic of the vegetable kingdom. But C. Schmidt† proved their existence in the mantle of the Phallusia, one of the Tunicata; Lœwig and Kölliker‡

* Ueber Speck-und cholesterinkrankheit. Annalen des charitékrankenhauses, Band vi. Quoted by Pagenstecher.

† C. Schmidt Zur vergleichenden Physiologie der wirbellosen Thiere, s. 62.

‡ Annales de Science Naturelle, 3mo Ser. Tom v., p. 193.

found a similar material in some Ascidians, and Dr Berthelot* succeeded in transforming this matter into sugar. Professor Purkinje of Prague, discovered in the brain and spinal cord the bodies which he termed corpora amylacea. They resemble starch granules in their appearance, being of oval form, and distinctly composed of concentric layers. Some of them assume a bluish colour with iodine; others a reddish hue, and, on the further addition of sulphuric acid, become blue or purple. Some again, which are incrusted with calcareous material, present no such reactions, at least, until the mineral matter has been dissolved by acids. Similar concretions occur in the prostate gland; and Friedreich† has described them as occurring in various morbid conditions in the lungs. All these corpora amylacea are in truth local products, consisting of successive layers deposited from the fluids of the tissues around a nucleus of blood or other matter. It was conceived that these materials might be truly starchy, or allied to starch; and Dr Carter‡ even stated that he had found starch granules in many of the healthy tissues of the body. Virchow,§ observing a certain starch-like appearance in the sago spleen, tested it with the re-agents which had been applied to the corpora amylacea, and thereby discovered the reaction above described. Advancing in the line of thought which was then followed by scientific men, he concluded that this degeneration might, in truth, consist in the production of a substance allied to cellulose, instead of the natural tissue of the affected part, and proposed for it the name amyloid degeneration. This term is now universally recognised as incorrect, but being in general use may be still retained. Carl Schmidt‖ attempted to form sugar from the waxy material and the corpora amylacea, but in vain.

* Récherches sur la transformation en sucre de divers principes immediats contenus dans les tissus des animaux invertébrés. Gazette Medicale, 1857, p. 618.

† Virchow's Archiv, Band ix., s. 613.

‡ Edinburgh Medical Journal, March 1858.

§ Virchow's Archiv, Band vi., and Cellular Pathology, p. 371.

‖ Annal. d. Chemie und Pharmacie, Band cx., s. 250.

Kekule and Friedreich* made a number of careful analyses of waxy spleens, and found that, though the waxy spleen contains a considerable amount of cholesterin, that substance is not the cause of the reaction with iodine and other reagents; that the waxy spleen contains no material chemically resembling starch or cellulose; and that the so called waxy material is closely allied to albumen and other members of the protein group of substances, for they found it to be—

$C_{53\cdot58}$ $H_{7\cdot00}$ $N_{15\cdot04}$

while albumen is, according to Dumas and Cahours—

$C_{53\cdot5}$ $H_{7\cdot1}$ $N_{15\cdot8}$
$C_{53\cdot4}$ $H_{7\cdot2}$ $N_{15\cdot7}$
$C_{53\cdot5}$ $H_{7\cdot3}$ $N_{15\cdot7}$

According to Lieberkühn—

$C_{53\cdot5}$ $H_{7\cdot0}$ $N_{15\cdot6}$

And according to Rüling—

$C_{53\cdot8}$ $H_{7\cdot1}$ $N_{15\cdot5}$

They thus made out the important fact that the waxy material is a nitrogenous substance closely allied to albumen.

Kuhne and Rudneff† repeated the experiments of Kekule and Friedreich, employing certain new methods, with the result of fully confirming their conclusions. They point out that the waxy material differs from ordinary albumen in several interesting particulars, for example in its slowness of decomposition, they kept pieces exposed to the air for months, and found that it did not alter, and in its resisting solvents, such as weak alkalies, not quite concentrated acids, and gastric juice.

Dr Dickinson‡ states, that from analysis of seven waxy livers compared with seven healthy organs, he found that while the latter contained 1 per cent of alkaline salts, the

* Virchow's Archiv, Band xvi., s. 59. † Virchow's Archiv, Band xxxiii., s. 66.
‡ Medico-Chirurgical Transactions, Vol. l., page 39.

former contained only ·754 per cent. He thus conceives that the waxy material differs from albumen and fibrine mainly in that it contains less alkali. I am not aware that any analyses have as yet been made to test the results obtained by Dr Dickinson; his statements upon this point must be recognised as interesting, though not in the meantime fully established.

Two main views are entertained as to the mode of production of the waxy material. Some, as Budd, Portal, Rindfleisch, and Dickinson, regard it as an infiltration or deposition of a peculiar matter from the blood; others, as Schrant, Rokitansky, and Oppolzer, regard it as a true degeneration or metamorphosis of tissue.

Rindfleisch* acknowledges that he cannot prove the existence of the material in blood, that he only knows it as it appears in the tissues. The evidence of its blood origin is, he remarks, merely circumstantial, and the only consideration he adduces is the observation of Friedreich and Biermer, that masses of extravasated blood assume, under certain conditions, the waxy reaction with iodine. He says that an albuminous material of the blood, passing through the walls of vessels, becomes consolidated, and constitutes the waxy material. As spontaneous consolidation is a characteristic of fibrine, he thinks it probable that the material is a modification of that substance. Dr Dickinson† has advanced a step further, and has sought to show that the exuded material is de-alkalized fibrine. His experiments have satisfied him that fibrine, artificially deprived of its alkali, reacts with iodine like the waxy material, that when alkali is restored to the fibrine, or added to the waxy material, the reaction no longer appears, and upon these data his theory is founded.

These views are interesting, and the observations, so far as I have seen, correct, but it is doubtful whether there are grounds for believing the substance derived from the blood at

* Lehrbuch der Pathologischen Gewebelehre, s. 33. † Loc. cit.

all. In seeking to solve the question, it is important to distinguish between the waxy degeneration proper and the secondary deposit of fibrinous material which results from it. That it is not an infiltration will become apparent if we consider the parts it affects. We have seen that it affects the cells of the liver. This might well be a result of infiltration, for we constantly see the same structures loaded with fat which has been poured out from the blood. But far more generally throughout the body it affects small arteries. How is this to be explained on the infiltration theory? How can it happen that the small arteries—nay, the small arteries of certain organs—nay, the middle coats of the small arteries—should be the chosen seat of deposit of a material poured out from the blood? If it be poured out from the blood, why does it so generally confine itself to the walls of arteries and unstriped muscular fibre? Why does it not infiltrate the parenchyma of organs? Why does it, in the intestine, as it flows from the blood, confine itself to the walls of small vessels, to the muscles of Brücke, to the epithelium, why does it not pass out among the cellular tissue of the villi and become deposited there? These questions can scarcely be answered by those who hold the infiltration theory.

On the other hand, how exactly the characters correspond to those of the degeneration,—certain tissues always primarily affected, those tissues presenting every degree of alteration, from the slightest to the most distinct, the surrounding parts remaining unaffected. The closest analogy exists between what is seen in this, and in the atheromatous and calcareous degenerations, the former affecting the inner coat of arteries, and especially the deeper layers, the latter, when primary, affecting the middle coat. Both are independent diseases originating in the tissues, not even secondary results of infiltration.

With regard to the cause of this degeneration, writers generally have been content to assign the process to the injurious results of syphilis, tuberculosis, caries, chronic sup-

puration, and other exhausting diseases; but Dr Dickinson has lately sought to show that it in truth depends upon only one of these causes,—viz., suppuration. This accomplished physician of course supports his theory with clinical evidence. He says, that "placing together the cases from both writers (Dr Wilks and myself), we have 109 in which the antecedent disease was traced; of these 83, or more than three-fourths, were consequent upon undoubted loss of pus, while in the remaining 26, the preceding disorders were of such a nature that it is not possible to doubt that at some period the same morbid discharges must have been present in the greater number." He also gives an account of 60 cases observed by himself. In 46 there was evidence of a profuse and long-continued drain of pus, in 4 there was reason to suspect that purulent discharges had occurred, of the remaining 10, 4 had suffered from severe albuminuria. So strongly is he of opinion that he has discovered the one true cause, that he proposes to substitute for the names at present in use one which he has invented, viz. "depurative." This term is singularly unfortunate, inasmuch as it necessarily suggests to the medical mind an idea very different from what is intended. For this reason alone it never could be popular; but the name is of little importance if the facts be correct. We now turn to that question.

It seems best to test the view by analysing a series of cases which I had myself observed, in which the existence of the waxy disease was proved by post-mortem examination, and whose antecedent history was carefully recorded, and for the accuracy of which I am personally responsible. I find 18 such cases, a small number, but quite sufficient to test the accuracy of such a theory as that under consideration. Of these cases, 7 certainly, and 1 probably were from syphilis, 4 were from tubercle of the lungs, 2 from caries of bone, 1 had suffered from chronic rheumatism, 1 was affected with cancer, and 2 stated that their illness had not been preceded by any particular disease. Of the syphilitic cases, none had suffered

from suppuration; indeed, only 6 of the 18 cases could be regarded as confirming Dr Dickinson's observations. This test is, in my opinion, crucial, for in all the cases I was minutely acquainted with the patients' history, and suppuration had occurred only in one-third of the whole number. I am thus compelled to doubt the correctness of Dr Dickinson's views on this particular, and to maintain the opinion more commonly held.

The points, then, that appear well established as to this affection are—1*st*, That it is a true degeneration or transformation of tissue, not an infiltration. 2*d*, That it consists of an albuminous material, probably deficient in alkali. 3*d*, That it results from long-continued exhausting diseases, such as syphilis, tuberculosis, caries, and chronic suppuration.

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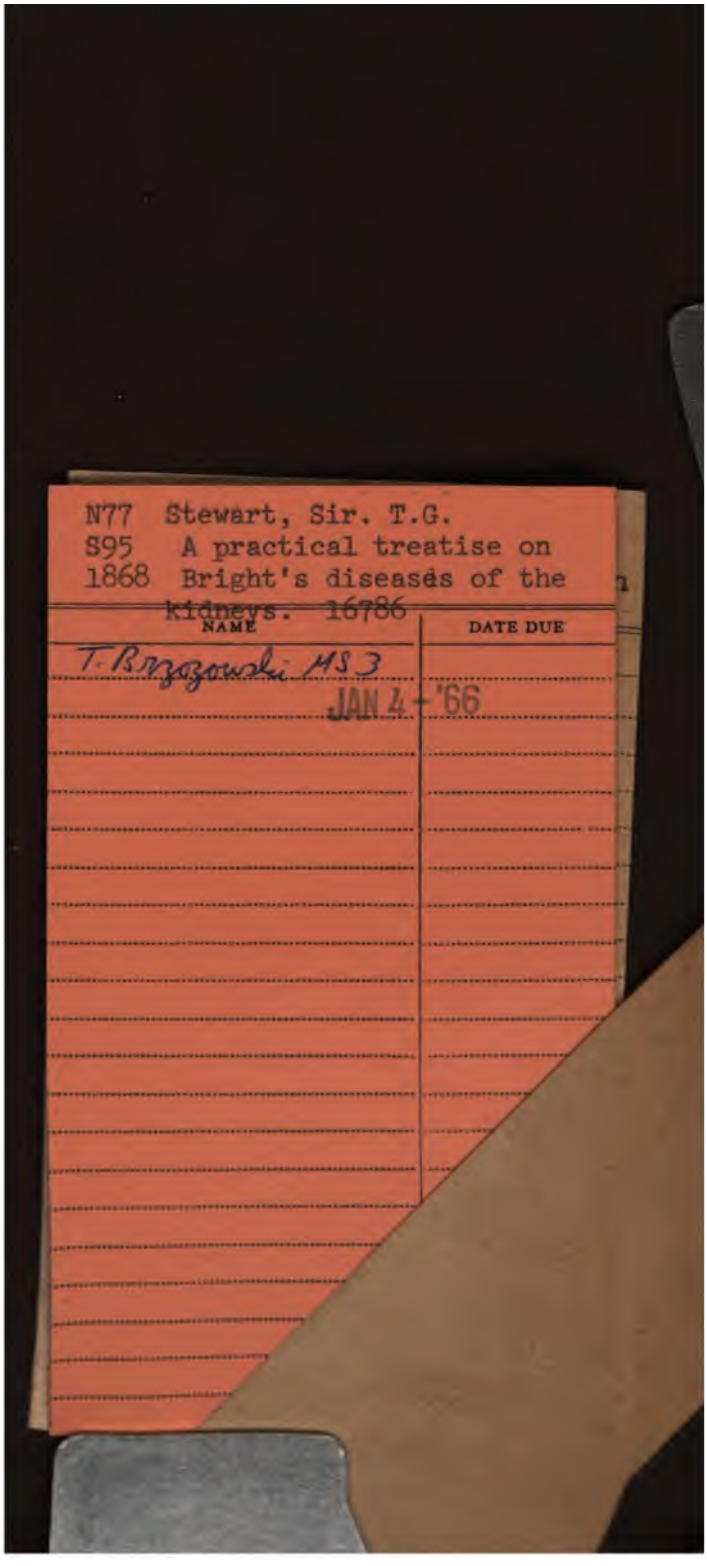
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